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<p>(54) Title: METHOD AND APPARATUS FOR DETERMINING ALVEOLAR OPENING AND CLOSING</p> <div data-bbox="422 1134 1169 1722"> </div> <p>(57) Abstract</p> <p>The invention refers to a method for the regional determination of the alveolar opening and alveolar closing of the lung depending on the respiration pressure, wherein according to the method of electrical impedance tomography, an impedance signal is measured in at least one lung zone depending on the respiration pressure. The alveolar opening or closing of a lung zone is determined, in particular to enable an improved artificial respiration.</p>		

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METHOD AND APPARATUS FOR DETERMINING ALVEOLAR OPENING AND CLOSING

5 The invention refers to a method for the determination of the alveolar opening and alveolar closing of the lung depending on the pressure respiration. In particular, the invention enables to a regional determination of the alveolar opening and alveolar closing.

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It is known that to measure the lung mechanics, pressure and volume should be recorded and superimposed. If one increases the pressure continuously, as from a certain pressure the first alveoli (terminal lung units or air sacks) begin to
15 change over from the state of collapse into the state of openness. If the pressure is increased further, more and more of the closed alveoli are opened. The maximum number of state changes takes place finally at the turning point of the pressure / volume curve. Thereafter, the opening ebbs away on
20 a further increase in pressure and changes over into saturation, wherein ideally all the alveoli are opened.

One problem in the measurement of the lung mechanics is that the distribution of this opening phenomenon is not
25 homogeneous over the entirety of the lung. For example, the lung is made heavier by the oedema formation, i.e. because of increased accumulation of liquid in the case of inflammations. Thereby a gravity dependent gradient results from the sternum to the spinal column. Thereby above all the
30 lowest parts of the lung are compressed and collapse.

In the case of a traditional pressure-volume measurement, however, one does not receive any information concerning the regional pressure-volume relationship, but one only receives
35 average information on the pressure-volume relationship of the entire lung.

For the regional measurement of the pressure-volume relationship the so-called electrical impedance tomography is

known. In this process, a number of electrodes are placed around the thorax, wherein an alternating current with e.g. 50 kHz at 5 nA peak to peak amplitude is applied to respectively adjacent electrodes. The other electrodes
5 respectively are used with the alternating current to carry out the measurement of impedance against a defined reference potential. As soon as all the electrodes, one after another, have served as current conducting electrodes, a cycle for data detection is concluded. In order to eliminate
10 statistical disturbances, as a rule a plurality of data detection cycles is averaged, in order to obtain a corresponding picture. The maximal impedance changes in the zone of the thorax are caused by the breathing in and out of air. In this context it can be observed that the impedance
15 change which is measured by electrodes is a measure of the change of volume in the lung. Therefore according to the process of electrical impedance tomography, measurements can also be carried out with respect to the pressure-volume relationship in the lung. However, the special feature of
20 electrical impedance tomography is that on the basis of a computer-based evaluation of the signals at the electrodes, a two-dimensional or even three-dimensional image of the impedance changes can be compiled.

25 The artificial respiration of a sick lung, wherein oedemas have formed, is a special problem, because it cannot be exactly controlled whether the lung has already closed and/or collapsed in certain parts. Then it was found that the mortality rate can be reduced substantially when a
30 predetermined pressure is artificially maintained in the lung, which just makes possible keeping open all the alveoli (terminal lung units, air sacks). However, this pressure is not known in the case of artificial respiration, because the alveolar opening and/or closing of the lung could not yet be
35 regionally determined.

Therefore the object of the invention is to make available a method for the determination of the alveolar opening and

alveolar closing of the lung, depending on the respiration pressure.

This object is solved by a method comprising the features according to claim 1 and by an apparatus comprising the features according to claim 25. The method according to the invention is based on the cognition that the alveolar opening and/or closing can be determined from an impedance signal gained with the method of electrical impedance tomography. Thereby at least two important values can be determined, namely a first respiration pressure value which corresponds to the alveolar closing of the corresponding lung zone and a second respiration pressure value which corresponds to the alveolar opening of the corresponding lung zone.

Accordingly, an apparatus for carrying out the method according to the invention comprises a plurality of electrodes which are applied around the thorax, an electrical impedance tomograph for the control of individual electrodes and for the evaluation of impedance signals on electrodes which are not controlled, in order to obtain a regional impedance signal in the thorax, and a processing unit to evaluate the regional impedance signals for determining the first respiration pressure value and the second respiration pressure value.

In contrast to computer tomography and magnetic resonance tomography, the process according to the invention can also be carried out at the bed of the patient, because no costly instruments are necessary. In this case there is no radiation stress either for the patient or for the staff. In the case of critical patients constant supervision of the state and degree of openness of the lung can therefore be carried out.

The first effect of the process according to the invention is that the impedance signal is influenced by the breathing movements of the patient. In each breathing movement the lung volume rises and falls. Using the regional impedance curves of electrical impedance tomography it can be observed that

the average change of the impedance signal, due to breathing movements, is conspicuously greater in zones wherein the lung has not yet collapsed, whereas in zones wherein the lung has already collapsed, only minor changes in the impedance signal are caused. For example the change in the impedance signal due to breathing movements can be determined on the basis of the unaveraged root mean square of the impedance signal over a plurality of breaths. The change in the impedance signal on the basis of breathing movements is therefore determined from the signal energy of the high frequency portions of the impedance signal, which are based on the breathing movements. But it is equally possible that the change in the impedance signal based on breathing movements can be determined on the basis of an average peak to peak value of the impedance signal over a plurality of breaths.

The alveolar closing and/or opening of the lung or the first and second respiration pressure value respectively is determined on the basis of the change in the impedance signal due to breathing movements, in that the change in the impedance signal based on breathing movements is compared with predetermined breathing movement comparative values. In doing so, it must be taken into account that with respect to the two comparative values, as a rule a certain hysteresis is found. This means that the opening of the pulmonary cells does not take place at the same pressure as the closing of the alveoli (terminal lung units), but that both comparative values fall away from each other. In this context it must in addition be taken into consideration in which direction the respective comparative value passes in order to be able to precisely identify the hysteresis.

With respect to the comparative values it is conceivable that fixed comparative values are predetermined. However, in this case disturbance factors, e.g. based on offset changes, enter fully into the measurement. Therefore it is expedient to determine the breathing movement comparative values dynamically from the average change in the impedance signal

on the basis of breathing movements of another zone of the lung. Preferably the lung is divided into a plurality of zone planes perpendicularly to the gravity vector, wherein the other lung zone is a zone which is in the direction of the gravity vector above the lung zone which is concerned. In this case use is made of the fact that as a rule the lung part which is lowest in the direction of the gravity vector is more strongly affected by the pathological appearance of the collapse of the alveoli (terminal lung units) than the correspondingly higher part of the lung zone. Alveolar closing of a lung zone, for example, can be determined as soon as the breathing movement comparative value of the lower lung zone is less by a predetermined factor than the breathing movement comparative value of the lower zone.

15 A further effect which is suitable to determine the alveolar opening or closing of the lung or the first and second respiration pressure value respectively is the change in the impedance signal due to the collapse of the alveoli. In the case of a pathological lung or an unphysiological condition such as i.e. anaesthesia it is observed that even with constant pressure the lung zone collapses, i.e. the pulmonary units therefore collapse spontaneously. This collapse takes place all the more strongly as the respiration pressure falls, wherein the effect in addition is reinforced like an avalanche over time. Consequently according to the invention alveolar closing of the lung zone or the first respiration pressure value respectively is determined as soon as the average change in the impedance signal due to the collapse of the alveoli falls below a collapse comparative value. Accordingly alveolar opening of a lung zone or the second respiration pressure value respectively is found as soon as the average change in the impedance signal based on the opening of the alveoli is above an opening comparative value.

35 The average change in the impedance signal due to the collapse of the alveoli, for example, can be determined on the basis of the mean increase in the impedance signal depending on time with a predetermined respiration pressure.

The average increase, for example, can be determined by the Gauß compensation computation, in that a straight line is placed in a segment of the impedance signal depending on time at constant pressure. The collapse comparative value and/or
5 the opening comparative value can be prescribed as fixed values, or however they can be determined from a dynamic comparative value determination. The dynamic determination of the comparative value is carried out expediently on the basis of an impedance signal in a different lung zone. Preferably
10 the lung is divided, as was described above, into a plurality of zone planes in the direction of the gravity vector, wherein the comparative value is derived from the lung zone which is above the lung zone concerned in the direction of the gravity vector.

15 A further effect caused by the alveolar opening or closing of a lung zone is the average change of the impedance signal on the basis of respiration pressure changes. As soon as a sudden respiration pressure change is applied to the lung,
20 the impedance signal for this pressure change does not follow at once, but respectively with a certain delay.

Accordingly, alveolar closing or the first respiration pressure value respectively of a lung zone is determined, as
25 soon as the average change in the impedance signal based on respiration pressure changes falls below a first respiration pressure comparative value, and wherein an alveolar opening or the second respiration pressure value respectively of a lung zone is determined as soon as the average change of the
30 impedance signal based on respiration pressure changes moves above a fixed second respiration change comparative value. In this context use is made of the observation that the lung mechanics responds with a certain inertia to changes in pressure. This inertia is larger in the sick zones than in
35 the healthy zones of the lung, because the sick zones only open as from a higher pressure, so that the sick zones can be localised according to the invention.

The change in the impedance signal due to respiration pressure changes, for example, can be determined on the basis of the average initial rise in the impedance signal after a sudden increase in respiration pressure. The initial
5 rise is all the smaller, the more the lung zone which is concerned tends on the basis of pathological changes to a collapse of the terminal lung units or alveoli. Another possibility is that the change of the impedance signal on the basis of respiration pressure changes is determined based on
10 the time constant of the impedance signal, with which the impedance signal follows a change in the respiration pressure. The first respiration pressure comparative value and/or the second respiration pressure comparative value can be prescribed or, however, can be determined dynamically, as
15 was described already above for the other processes. In the case of dynamic determination of the first respiration pressure comparative value and/or of the second respiration pressure comparative value, the determination is carried out on the basis of the average change of the impedance signal
20 due to respiration pressure changes in another lung zone. The other lung zone is again preferably a zone which is in the direction of the gravity vector above the lung zone concerned. In this process the lung is subdivided for the measurement into a plurality of zone planes in the direction
25 of the gravity vector.

According to a preferred embodiment it is provided that setting out from a respiration pressure wherein the lung alveoli are opened in almost all the lung zones, the
30 respiration pressure is reduced step by step, until an alveolar closing of a lung zone is found in one lung zone.

Apart from the division of the lung into zones in the direction of the gravity vector, it is also conceivable that
35 the lung is divided into a plurality of radial sectors, wherein the centre point axis of the sectors is in the direction of the gravity vector.

A device for carrying out the method according to the invention consists of a plurality of electrodes which are applied around the thorax, of an electrical impedance tomograph for the control of individual electrodes and for the evaluation of the impedance signals at the uncontrolled electrodes, in order to obtain a regional impedance signal in the thorax, and of a processing unit to evaluate the regional impedance signals for determining the first respiration pressure value and the second respiration pressure value.

10 Falsification of the signals is to be determined in this context, in particular, due to breathing movements, because on each intake or outlet of breath, the positions of the electrodes in relation to each other alter. In order to eliminate the resultant signal falsifications at the electrodes, a sensor is provided to measure the changing periphery of the thorax caused by the breathing movements. In addition, the electric impedance tomograph comprises a correction unit, wherein the change of impedance signals of the electrodes caused by breathing movements is corrected by

20 including the sensor signal.

An important aspect of the apparatus according to the invention is to control an artificial respiration unit. This can be particularly useful for a sick lung because it cannot be exactly controlled whether the lung has already closed and/or collapsed in certain parts. However, according to the invention it was found that the mortality rate can be reduced substantially when a predetermined pressure is artificially maintained in the lung, which just makes it possible to keep open all the alveoli. This can be done by providing a control unit which is connected to the artificial respiration unit and the processing unit, whereby the first respiration pressure value and the second respiration pressure value is fed from the processing unit to the control unit to control

35 the artificial respiration.

The signals obtained by regional impedance tomography can be used to determine an optimal therapeutic level of the so-called positive end-expiratory pressure (PEEP). It is

important to find an optimal biological compromise between treating alveolar overdistension in one part of the lung and atelectasis in another. As a priority, PEEP levels must be set high enough to prevent as much as possible the collapse of alveoli at the end of expiration in the most dependent parts of the lung; at the same time the over-stretching of the non-dependent upper parts on the lungs must be avoided. Both these pathological conditions -alveolar collapse and alveolar overdistension - can be recognized as a reduced amplitude of the ventilation-induced impedance changes in a regions of interest. An optimal level of PEEP, however, leads to an even distribution of ventilation (and thus impedance changes) throughout the entire lung.

15 In addition, an optimal level of PEEP prevents the collapse of airways. If airways are kept open during the entire respiratory cycle, the respiratory gases are exchanged efficiently. These parts are thus ventilated and the impedance signals follow this ventilation. If, however, the conducting airways are collapsed during the entire respiratory cycle, the terminal lung units -in particular the alveoli- are cut off from the supply of fresh gas. Gas exchange suffers and no ventilation-induced change in the impedance signal can be detected. These lung areas become silent on the impedance tomographic image. The oxygen within the cut-off alveoli is absorbed and with the progressive decrease in their gas content, the absolute impedance of such a lung unit is reduced. In a scenario where PEEP levels are not high enough to prevent the expiratory collapse of airways and terminal lung units (alveoli) but where pressures are sufficiently high to open collapsed airways during inspiration, ventilation of these lung units takes place only during this period of the respiratory cycle. The changes in the impedance signals of such a lung region can be amplified compared to an area of normal ventilation since these collapsed lung units start from a low expiratory air content but are filled rapidly to approximately normal volumes during inspiration. During expiration they collapse, again and the process of tidal recruitment/collapse begins anew.

Observing the signals from regional impedance tomography it is possible to determine the points of airway/alveolar opening and closing by systematically titration inspiratory and expiratory airway pressures.

In accordance with a further aspect of the present invention, the apparatus comprises a monitoring unit for monitoring the first respiration pressure value and the second respiration pressure value. By monitoring these values the patient can be observed by a monitoring device gaining important pieces of information with regard to the lung functioning. All the direct and derived impedance signals and/or images discussed above should be calculated continuously and should be available for on-line display. Any single one of them or a combination of them can be used for the automatic or semi-automatic control of a therapeutic device, such as a mechanical ventilator. The information obtained by electrical impedance tomography can be used to guide specific clinical maneuvers aiming at optimal lung recruitment and at keeping most alveoli open or at finding the best biological compromise between alveolar over-distension and alveolar collapse.

Furthermore, regional pressure-volume curves generated by electrical impedance tomography can be used to define pressure points of specific clinical relevance. These points are the alveolar opening and closing pressure of a specific lung region, the lower and the upper inflection point of the inspiratory and the expiratory pressure-volume curve. Additional information on lung behavior can be obtained by analyzing the shape and the area the pressure-volume-curve.

Further details and advantages of the invention will be explained in more detail on the basis of the example of an embodiment shown in the drawing. It shows:

Fig. 1 pressure-impedance curves in four different zones of the lung,

- Fig. 2a an impedance signal depending on time for the entire lung,
- 5 Fig. 2b an impedance signal depending on time for the upper zone of the lung,
- Fig. 2c an impedance signal depending on time for the lower lung zone with the relevant pressure curve for
10 figures 2a, 2b and 2c,
- Fig. 3a an impedance signal depending on time for the entire lung zone,
- 15 Fig. 3b an impedance signal depending on time for the upper lung zone, and
- Fig. 3c an impedance signal depending on time for the lower lung zone with the relevant pressure signal for
20 figures 3a, 3b and 3c,
- Fig. 4 a superimposition of a pressure-impedance and a pressure-volume curve of an entire lung during inflation and deflation,
25
- Fig. 5 three curves indicating the changes of impedance during mechanical ventilation as a function of time,
- 30 Fig. 6 impedance signals of the upper and the lower parts of the lung together with the signal of the total lung during a slow insufflation at a constant flow of oxygen,
- 35 Fig. 7 independent inflation-deflation pressure-impedance curves of the upper and the lower part of the lung,

- Fig. 8 impedance curves of the upper and lower parts of the lung at decreasing levels of positive end-expiratory pressures (PEEP),
- 5 Fig. 9 impedance curves of the upper and the lower lung of a patient suffering from severe lung failure, and
- Fig. 10 impedance curves according to Fig. 9 together with an arterial oxygenation index,
- 10 Fig. 11 an external electrodes set up,
- Fig. 12 an internal electrodes set up,
- 15 Fig. 13 an electrical impedance tomography internal and external electrodes set up,
- Fig. 14 shows a electrical impedance tomography set up with internal electrodes using an intratracheal catheter, an esophageal catheter, a pulmonary artery catheter and a superior vena cava catheter,
- 20 Fig. 15 shows a superior vena cavae internal electrode set up,
- 25 Fig. 16 shows a pulmonary artery (swan-ganz) internal electrode set up,
- Fig. 17 shows an intra-tracheal tube internal electrode set up and
- 30 Fig. 18 shows an esophageal tube internal electrode set up.

Figure 1 shows pressure-impedance curves according to electrical impedance tomography in four different zones of the lung. In comparison with the known pressure-volume curves, the corresponding pressure-impedance curves show a similar course. As from a certain pressure point, the first alveoli (terminal lung units or air sacks) change over from

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the state of collapse to the state of opening. When the pressure is further increased, more and more closed alveoli are opened until the opening finally ebbs away and at higher pressures forms the flat part of the impedance signal.

- 5 Comparison of the individual curves over the various zones of the lung shows that the opening phenomenon is not homogeneously distributed over the entire lung in this case. The measurements are carried out according to the method of electrical impedance tomography, wherein the zones 1 to 4 in
10 the direction of the gravity vector subdivide the lung into planes which are perpendicular thereto. In the uppermost zone of the lung, the expected pressure-impedance distribution appears, whereas in the regions 2 to 4, increasingly pathological manifestations of the closing phenomenon are
15 seen to be recognized. For example, pathological changes in the lung may be caused by oedema formation (increased accumulation of liquid in the case of inflammation), whereby the lung is heavier in the direction of the gravity vector. Inter alia, above all the lowest parts of the lung are
20 compressed thereby and therefore can only open at a later point in time or at higher pressures.

- Figures 2a, 2b and 2c show impedance signals depending on time for different zones of the lung, wherein as the pressure
25 signal, the pressure signal marked in fig. 2c respectively forms the basis. After one half of the paths of the curve, there is respectively a change in the scale, wherein in the second half of the figures, the path of the curve is correspondingly compressed. Figure 2a shows an impedance
30 curve for the total zone of the lung, whereas the path of the curve according to fig. 2b concerns the upper zone and the path of the curve according to fig. 2c refers to the lower zone of the lung. In fig. 2c the underlying pressure signal is marked, which refers to all three figures. Accordingly the
35 respiration pressure is suddenly increased after a certain initial time and then it is reduced step by step, until another pulse follows. The lower zone of the lung is in its turn pathologically altered. According to the invention, this

pathological alteration can be discerned in the curves which are shown, in particular, on the basis of two processes:

On the one hand it is possible to evaluate the change in the impedance signal due to breathing movements. They are expressed in the impedance signal in high frequency oscillations, the sinusoid course of which is to be discerned in the first half of the signals. When one compares the changes in the impedance signal on the basis of breathing movements according to the parameters A_1 and A_1' it is noticeable that the breathing movements in the upper zone of the lung cause larger impedance changes than in the lower zone of the lung. In addition it is striking that this phenomenon is dependent on respiration pressure, as a comparison of the magnitudes A_2 and A_2' shows.

Another process according to the invention for the regional determination of alveolar opening and closing of the lung consists of the evaluation of the mean change in the impedance signal based on the collapse of the alveoli. This effect is marked in figures 2b and 2c by the magnitudes B and/or B' . The impedance signal according to fig. 2b fluctuates at constant pressure around a constant offset, whereas in the impedance signal according to fig. 2c, a drop in the impedance signal is also to be seen at constant pressure. Consequently the ascending gradient B and/or B' makes a statement as to whether collapse of the lung is taking place:

Figures 3a, 3b and 3c show an impedance signal as the response to a pulse-shaped pressure increase, which is shown in fig. 3c. In the lower zone of the lung according to fig. 3c, the pulse signal responds thereto with a delayed response, whereas the impedance signal according to fig. 3b follows the pressure increase without delay. Therefore a method for regional determination of the alveolar opening and closing of the lung can be derived from the change in the impedance signal on the basis of respiration pressure

changes. For example, this change can be inferred from the initial gradient of the impedance signal on pressure changes.

- 5 Another possibility is to analyse the phase difference during conventional tidal breaths between different lung zones. Having two wave forms of tidal breaths of the impedance signal, one from the upper level and one from the bottom level, the change in the impedance signal on the basis of
10 respiration pressure changes can be calculated from the phase difference between these two sinusoidal-like curves. This kind of analysis showed also very consistent results.

- In summary, there are at least three possibilities to
15 determine the alveolar opening and the alveolar closing of the lung from the impedance signal: Firstly, regional amplitudes detected as the distance between peaks and valleys during tidal breaths or just as the standard deviation of the signal during a certain period of time can be analysed,
20 either for one region or as a comparative method for different regions. Secondly, knowledge-based methods can be introduced as shown e.g. according to Fig. 2c where the impedance curve shows a behaviour which differs from the expected behaviour of a healthy lung. Furthermore, it can be
25 use of temporal delays of inflation of the impedance signal, either in one region or among different regions.

- Figures 4 to 10 show additional impedance curves of a patient with a sick lung. As described above, the lung is made
30 heavier by the oedema formation, i.e. because of increased accumulation of liquid in the case of inflammations. Thereby a gravity depend gradient results from the sternum to the spinal column. Thereby above all the lowest parts of the lung are compressed and collapse.

- 35 Fig. 4 shows a superimposition of a pressure-impedance and a pressure-volume curve of an entire lung during inflation and deflation.

Fig. 5 shows three curves indicating the changes of impedance during mechanical ventilation as a function of time. The uppermost curve represents the upper, the lowest curve the lower part of the lung. The middle curve represents the impedance changes of the entire lung (upper and lower parts together). After an initial phase of steady state, ventilation is stopped. The lungs collapse immediately (they de-recruit). Then, the lungs are inflated with a constant flow of breathing gas. Note the delay in time (indicated by the arrow) before the impedance of the lower part of the lung begins to show a positive change in its impedance signal. Thus, a considerable time lag in the recruitment of alveoli in the lower, most dependent part of the lung is noticed. After the successful recruitment manoeuvre, a new steady state of ventilation is reached. Now, the amplitude of the signal and the mean level of impedance in the lower part have both increased.

Fig. 6 shows impedance signals of the upper and the lower parts of the lung together with the signal of the total lung during a slow insufflation at a constant flow of oxygen. The upward convexity of the upper curve indicates a distension of open alveoli as lung volume increases. The upward concavity of the curve representing the lower lung areas indicates a delayed (arrow) opening of collapsed lung units. The steep slope of the curve beyond 90seconds shows that the recruitment process is still going on without ever reaching a saturation as in the upper lung. As can be expected from the experimental set up the curve of the total lung is almost a straight line; it represents the change in the air content of the total lung. It increased linearly with time.

Fig. 7 shows independent inflation-deflation pressure-impedance curves of the upper and the lower part of the lung. Compared to the upper curve the lower curve is shifted towards the right, indicating a delayed opening of dependent alveoli. As opposed to the upper lung, the lower one does not show a saturation behaviour of its impedance changes at high away pressures. Thus, in the dependent lung zones the

recruitment of collapsed alveoli still incomplete even at airway pressures as high as 50 cmH₂O. On the deflation limb, when airway pressures are reduced, collapse of the lower lung regions occurs earlier than in the respective upper lung zones (arrows indicate alveolar closing).

Fig. 8 shows impedance curves of the upper and lower parts of the lung at decreasing levels of positive end-expiratory pressures (PEEP). The impedance amplitude of the upper lung (U) is divided by the amplitude of the lower (L) lung. The U/L-ratio is given in the top line. With decreases in PEEP the mean impedance of these lung units decreases, too. At high PEEP levels the upper lung zones are distended (small amplitude) and ventilation is shifted to the lower lung zones (large amplitude). The U/L ratio remains below 1. Once overdistension is relieved, ventilation is distributed more evenly ($U/L \sim 1$). Once PEEP becomes too low to keep all lung units open, alveoli start to collapse. The amplitude of the impedance signal of the lower lung decreases and shifts to the upper lung regions. The U/L ratio exceeds 1. Finally, hardly any ventilation-induced impedance change can be seen in the lower curve.

Fig. 9 shows impedance curves of the upper and the lower lung of a patient suffering from severe lung failure (adult respiratory distress syndrome, ARDS) on day one on mechanical ventilation. PEEP is stepwise decreased from 12 to 0 cmH₂O. Initially, as distension is overcome, the amplitude of the impedance in the upper lung zones increases at the expense of the ventilation of the respective lower lung zones. Finally, at a PEEP level below 4 cmH₂O a decrease in the impedance amplitude indicates that alveolar collapse has also occurred in the upper lung zones. When, after this collapse, the PEEP level is returned to its original level (100%), the lung zones do not reach their original state of inflation, again. Despite the same distending pressure, the upper part achieves 80%, the lower lung only 42% of its original impedance (thus volume).

Fig. 10 shows two curves which are the same as before. In addition, arterial oxygenation index ($\text{PaO}_2/\text{FiO}_2$) is shown in the lower line. The open lung is characterized by a $\text{PaO}_2/\text{FiO}_2 > 500$ mmHg. As PEEP is decreased, the lower lung units start to collapse and ventilation is shifted towards the upper lung zones. This way, the loss of gas exchanging alveoli in the lower part of the lung is at least partially compensated. Oxygenation index decreased only slowly. Once, however, the PEEP is no longer high enough to stabilize the upper lung zones, their collapse is indicated by a steep drop in oxygenation curve. Even when setting the PEEP back to the original value, the loss of functional lung units is not reversed. Only 52% of the baseline oxygenation can be achieved.

As already mentioned above, the invention can make use of an electrical impedance tomography apparatus. However, it has to be observed that several adoptions and variations of the conventional electrical impedance tomography apparatus are possible to optimise the measurement according to the invention. This optimisations are described in the following with reference to the figures 11 to 18.

Fig. 11 shows an optimised external electrodes set up according to the invention. In order to overcome the known contact problems of conventional skin electrodes (high resistance to electrical currents, poor contact between skin and electrode, displacement and electrical noise with motion and breathing, etc) electrical bobbins to generate and detect magnetic field could be used. These could be arranged on circular band around the thorax or on catheters within the body. Alternatively the bobbins could be mounted on a fixed frame that encompasses the thorax. This frame could then be moved relative to the longitudinal direction of the body to obtain tomographic or spiral images of different segments of the thorax.

Furthermore, it should be noted that the number of electrodes can be increased from 16 to 32 or more electrodes in order to

improve the resolution of the signal obtained by regional electrical impedance tomography even more.

Fig. 12 shows an internal electrodes set up according to the invention. Generally speaking, the set up according to Fig. 12 is based on the cognition that the distance between the electrodes should be reduced. It is conceivable that electrodes or bobbins could be mounted on tubes and catheters that are placed within the body. Since both the trachea and the esophagus are located in the approximate centre of the thorax endotracheal and/or naso-gastric tubes could be used as electrical centres for the generation of regional electrical impedance tomographic images. Furthermore, catheters brought into the blood stream, such as central venous or pulmonary artery catheters could serve a similar purpose. Bobbins or electrodes could be placed on one single or on multiple locations along the tubes and/or catheters in order to obtain images at different locations within the chest. It could be feasible to use one or more of these tubes and/or catheters at the same time. Depending on the clinical situation of the patient, tomographic images of the electrical impedance of the chest can thus be generated by using external electrodes/bobbins around the thorax alone or by combining them with internal electrodes/bobbins as described above. Any one of the catheters or tubes has to be designed according to the needs defined by its general clinical purpose and by its specific function within the impedance tomography setting.

Fig. 13 and 14 show a set up in which all electrodes of the internal set up according to Fig. 12 are used for electrical impedance tomography measurements. As it becomes from Fig. 14, the distances between the electrodes can be reduced significantly.

Images and signals from regional electrical impedance tomography can be used to detect clinically important and dangerous situations instantaneously. If the endotracheal tube is placed in the correct anatomical position within the

trachea, both lungs are ventilated evenly. If, however, the tube is advanced too far only one of the two main bronchi is intubated; thus only this one lung is ventilated. The EIT-signal for the non-ventilated lung will be electrically
5 silent whereas the other half of the lung shows a normal or an increased intensity.

To detect this condition, the regional impedance signal of a representative part of each lung has to be determined. If the
10 ventilation-induced impedance change falls below an expected reference value a high suspicion for the presence of an incorrect intubation is generated. In the presence of such a suspicion the magnitude of the local impedance change of the right has to be compared with that of the left lung. If the
15 difference exceeds a certain threshold, a one-sided intubation can be diagnosed with certainty.

If -for whatever reason- lung tissue is disrupted and free air gets into the space between the lung and the rib cage
20 (pneumothorax) or in a spaces within the lung (bulla), this pathological accumulation of air will, after an initial increase in local impedance, show a markedly reduced or no further change in its impedance. This region will become ``silent'' on the EIT-image. The cyclic ventilation of the
25 surrounding lung tissue demarcates the pneumothorax or bulla. A similar but opposite change in the impedance properties (a reduction) can be seen if fluid accumulated in the space between the lung and the rib cage (pleural effusion). Again the ventilated lung tissue demarcates the pathological fluid
30 accumulation.

Fig. 15 shows a set up where only the superior vena cavae is used for an internal electrode set up. Accordingly, Fig. 16 shows a pulmonary artery (swan-ganz) internal electrode set
35 up. Furthermore, according to Fig. 17, the intra-tracheal tube is used for an internal electrode set up. Eventually, according to Fig. 18, the esophageal is used for an internal electrode set up. Intrapulmonary, intra-abdominal and esophageal pressures can be measured by the appropriate tubes

or catheters (i.e. endotracheal, esophageal or gastric tubes, urine or intra-abdominal catheters). Each one of these pressures, a combination of them or a difference between them can be plotted against the signal from regional impedance tomography to obtain information about the regional pressure impedance relationship. During mechanical ventilation this information could be used to titrate the appropriate levels of airway pressure (i.e. peak or mean airway pressure or positive end-expiratory pressure) with respect to regional or global lung expansion and ambient, intra-abdominal, intra-thoracic or other pressures. Pressure and impedance signals should be fed into the same device.

In the following, several measures for the improvement of the signal quality will be described. The improvements in the efficiency and performance of the electrodes and the signal transmission will ameliorate the EIT image acquisition in terms of speed and reliability. This will allow obtaining the EIT data in synchrony with the respiratory cycle. The synchronization can be achieved using external ventilator signals, automated plethysmograph signals or with the system's own impedance signals. This is of physiological importance, as it will provide information about the regional lung changes along the respiratory cycle especially at end inspiration and expiration. This way tidal recruitment and de-recruitment of terminal lung (alveoli) within one respiratory cycle can be detected.

Furthermore the EIT image acquisition can also be triggered by or synchronized with the cardiac cycle using the signal from simple ECG electrodes. Regional changes in pulmonary perfusion can thus be analyzed. Furthermore the synchronization with the cardiac cycle will help reduce or eliminate cardiac disturbances of impedance images of the lung; the resolution of respiratory imaging will thus increase.

Today, electrical impedance signals of the thorax are relative signals (they reflect changes but no absolute

values) and it has been difficult to convert them into absolute numbers. Using the above mentioned catheters and/or tubes within the thorax it is conceivable that internal reference signals for electrical impedance (i.e. a tissue calibration factor) could be generated by currents that are injected and/or received between two or more of these catheters or tubes.

The circumference of the thorax and therefore the distance between adjacent electrodes changes with breathing. These changes can easily be measured by conventional methods or detected automatically by plethysmographic means. Data reflecting these changes in circumference can be used within the algorithms for image reconstruction, thereby enhancing the quality of the impedance tomographic images. These data can either be inputted continuously or at discrete time intervals.

The quality of the images obtained by impedance tomography alone can be enhanced further if the data from morphometric measurements or anatomical images are superimposed. Ideally, measurements or pictures from computed tomography or magnetic resonance imaging are projected (mathematically, geometrically or literally) on top of the images obtained from impedance measurements. Areas with a certain electrical behavior can thus be seen in relation to their underlying anatomical structures. This way the size of "gray" zones with undetermined morphology and functionality can be reduced (i.e. areas of collapsed lung tissue could be distinguished from the rib cage, from intrapleural fluid or from bone, muscle or fat). Alternatively simple body measurements, (i.e. weight, height, body mass index, circumferences or others) could be used to normalize the mathematical algorithms for impedance image reconstruction.

In the following, an appropriate use of the regional impedance tomography is described to optimize airway pressure application in chronic obstructive pulmonary disease (COPD). In COPD the lung tissue loses its elastic recoil and

intrinsic stability. During expiration, small airways collapse if the pressure within them gets lower than a certain threshold pressure. Gas is thus trapped within the lungs. If inspiratory pressures are higher than the pressures required to re-expanding these collapsed airways, gas can move into the terminal parts of the lung and the alveoli. If the inspired amount of gas is larger than the amount that leaves the lung during expiration the lung is gradually expanded until a new steady state at high lung volumes is reached. The way the diseased lung tissue is easily overdistended and is rendered incapable of gas exchange.

In COPD the collapse of airways can be found in one part of the lung and the overdistension of lung units in another. Thus both these pathological situations can found at the same time.

At times, patients with COPD require support of their ventilation by the application of positive (or more infrequently negative) pressure ventilators. If the absolute amount of airway pressure is too high, lung tissue gets overdistended and dysfunctional for gas exchange. If, however, the applied pressures are too low to prevent the collapse of small airways, gas is trapped within the lung without being efficiently exchanged. Often, airway collapse and overdistension coexist within the same lung at a chosen pressure. For an optimal therapeutic result, the best compromise between these two conflicting lung conditions has to be found. Traditional lung mechanics give only a rough estimate of such a compromise. Information about the regional expansion and movement of air is required to approach this comprise.

Regional electrical impedance tomography provides data and images of regional lung ventilation. With increases in airway pressures the gradual emptying of trapped gas can be detected in one area of the lung, whereas other parts of the lung get progressively distended until in the truly overdistended stage no changes in impedance can be detected. By comparing

and integrating the quantities of overdistension and
emptying of the various portions of the lung at changing
airway pressures a best therapeutic "compromise pressure" can
be found that reflects optimal lung expansion at minimal
5 pressures.

Furthermore, not only electrodes can be used on the
catheters, but only the pressure measurements of the
catheters can be used for optimising the accuracy of the
10 regional pressure impedance curves.

Patent claims

1. Method for determination of the alveolar opening and alveolar closing of the lung, comprising the steps of:
 - 5 measuring according to the method of electrical impedance tomography an impedance signal (AU) in at least one lung zone depending on the respiration pressure,
 - 10 determining from the impedance signal a first respiration pressure value which corresponds to the alveolar closing of the corresponding lung zone, and
 - 15 determining from the impedance signal a second respiration pressure value which corresponds to the alveolar opening of the corresponding lung zone.
2. Method according to claim 1, wherein for the
 - 20 corresponding lung zone the first respiration pressure value is found as soon as the mean change in the impedance signal based on breathing movements (A_1 , A_2 , A_1' , A_2') falls below a first breathing movement comparative value and wherein the second respiration
 - 25 pressure value is found as soon as the mean change in the impedance signal due to breathing movements (A_1 , A_2 , A_1' , A_2') moves above a fixed second breathing movement comparative value.
3. Method according to claim 2, wherein based on a
 - 30 respiration pressure, with which the lung alveoli are opened in almost all the lung zones, the respiration pressure is reduced step by step, until in one lung zone alveolar closing of a lung zone is determined.
 - 35
4. Method according to one of the claims 2 to 3, wherein the mean change of the impedance signal due to breathing movements (A_1 , A_2 , A_1' , A_2') is determined based on the

unaveraged mean square root of the impedance signal over a plurality of inspirations.

5. Method according to one of the claims 2 to 3, wherein
5 the mean change in the impedance signal due to breathing movements is determined on the basis of an average peak to peak value of the impedance signal over a plurality of inspirations.
- 10 6. Method according to one of the claims 2 to 5, wherein the first breathing movement comparative value and/or the second breathing movement comparative value are predetermined.
- 15 7. Method according to one of the claims 2 to 5, wherein the first breathing movement comparative value and/or the second breathing movement comparative value are determined dynamically from the mean change in the impedance signal due to breathing movements in a
20 different lung zone.
8. Method according to claim 7, wherein the other lung zone is a zone which is above the lung zone concerned in the direction of the gravity vector.
25
9. Method according to claim 1, wherein for the corresponding lung zone the first respiration pressure value is found as soon as the average change in the impedance signal due to the opening/collapse of the
30 alveoli (B, B') falls below a collapse comparative value and wherein the second respiration pressure value is found as soon as the average change in the impedance signal due to the opening/collapse of the alveoli (B, B') moves above an opening comparative value.
35
10. Method according to claim 9, wherein setting out from a respiration pressure wherein the lung alveoli are almost all open in one lung zone, the respiration pressure is reduced step by step, until in the lung zone an alveolar

5 closing is found and in that setting out from a respiration pressure, wherein the lung alveoli in one lung zone are almost all closed, the respiration pressure is increased step by step until an alveolar opening is found in the lung zone.

10 11. Method according to one of the claims 9 to 10, wherein the average change in the impedance signal due to the collapse/opening of the alveoli (B, B') is determined on the basis of the average gradient of the impedance signal depending on the respiration pressure.

15 12. Method according to one of the claims 9 to 10, wherein the average change in the impedance signal due to the collapse/opening of the alveoli (B, B') is determined on the basis of a straight line adaptation according to the Gauß compensation calculation.

20 13. Method according to one of the claims 9 to 12, wherein the collapse comparative value and/or the opening comparative value are predetermined.

25 14. Method according to one of the claims 9 to 12, wherein the collapse comparative value and/or the opening comparative value are determined dynamically from the average change in the impedance signal due to the collapse of the alveoli in another lung zone.

30 15. Method according to claim 14, wherein the other lung zone is a zone which is above the lung zone concerned in the direction of the gravity vector.

35 16. Method according to claim 1, wherein for a corresponding lung zone the first respiration pressure value is found as soon as the average change in the impedance signal due to respiration pressure changes (C, C') falls below a first respiration comparative value and wherein the second respiration pressure value is found, as soon as the average change in the impedance signal due to

respiration pressure changes (C, C') moves above a fixed second respiration pressure comparative value.

- 5 17. Method according to claim 16, wherein on the basis of a respiration pressure at which the lung alveoli are open in almost all lung zones, the respiration pressure is reduced step by step, until in one lung zone an alveolar closing of a lung zone is found.
- 10 18. Method according to one of the claims 16 to 17, wherein the change in the impedance signal due to respiration pressure changes (C, C') is determined on the basis of the average initial gradient of the impedance signal after a sudden increase in respiration pressure.
- 15 19. Method according to one of the claims 16 to 17, wherein the change in the impedance signal due to respiration pressure changes (C, C') is determined on the basis of the time constant with which the impedance signal follows a change in respiration pressure.
- 20 20. Method according to one of the claims 16 to 19, wherein the first respiration pressure comparative value and/or the second respiration pressure comparative value are prescribed.
- 25 21. Method according to one of the claims 16 to 19, wherein the first respiration pressure comparative value and/or the second respiration pressure comparative value is/are determined dynamically from the average change in the impedance signal due to respiration pressure changes in another lung zone.
- 30 22. Method according to claim 21, wherein the other lung zone is a zone which is above the lung zone concerned in the direction of the gravity vector.
- 35

23. Method according to one of the claims 1 to 22, wherein the lung is subdivided into a plurality of zone planes in the direction of the gravity vector.
- 5 24. Method according to one of the claims 1 to 22, wherein the lung is divided into a plurality of radial sectors, wherein the centre point axis of the sectors is located in the direction of the gravity vector.
- 10 25. Apparatus for carrying out the method according to one of claims 1 to 24, comprising:
- a plurality of electrodes which are applied around the thorax,
- 15 an electrical impedance tomograph for the control of individual electrodes and for the evaluation of impedance signals on electrodes which are not controlled, in order to obtain a regional impedance
- 20 signal in the thorax, and
- a processing unit to evaluate the regional impedance signals for determining the first respiration pressure value and the second respiration pressure value.
- 25 26. Apparatus according to claim 25, wherein a sensor is provided for the measurement of the changing periphery of the thorax on the basis of breathing movements and in that the electrical impedance tomograph has a correction
- 30 unit, wherein the change in the impedance signals of the electrodes are corrected on the basis of breathing movements by including the sensor signal.
27. Apparatus according to claim 25 or 26, further
- 35 comprising:
- an artificial respiration unit, and

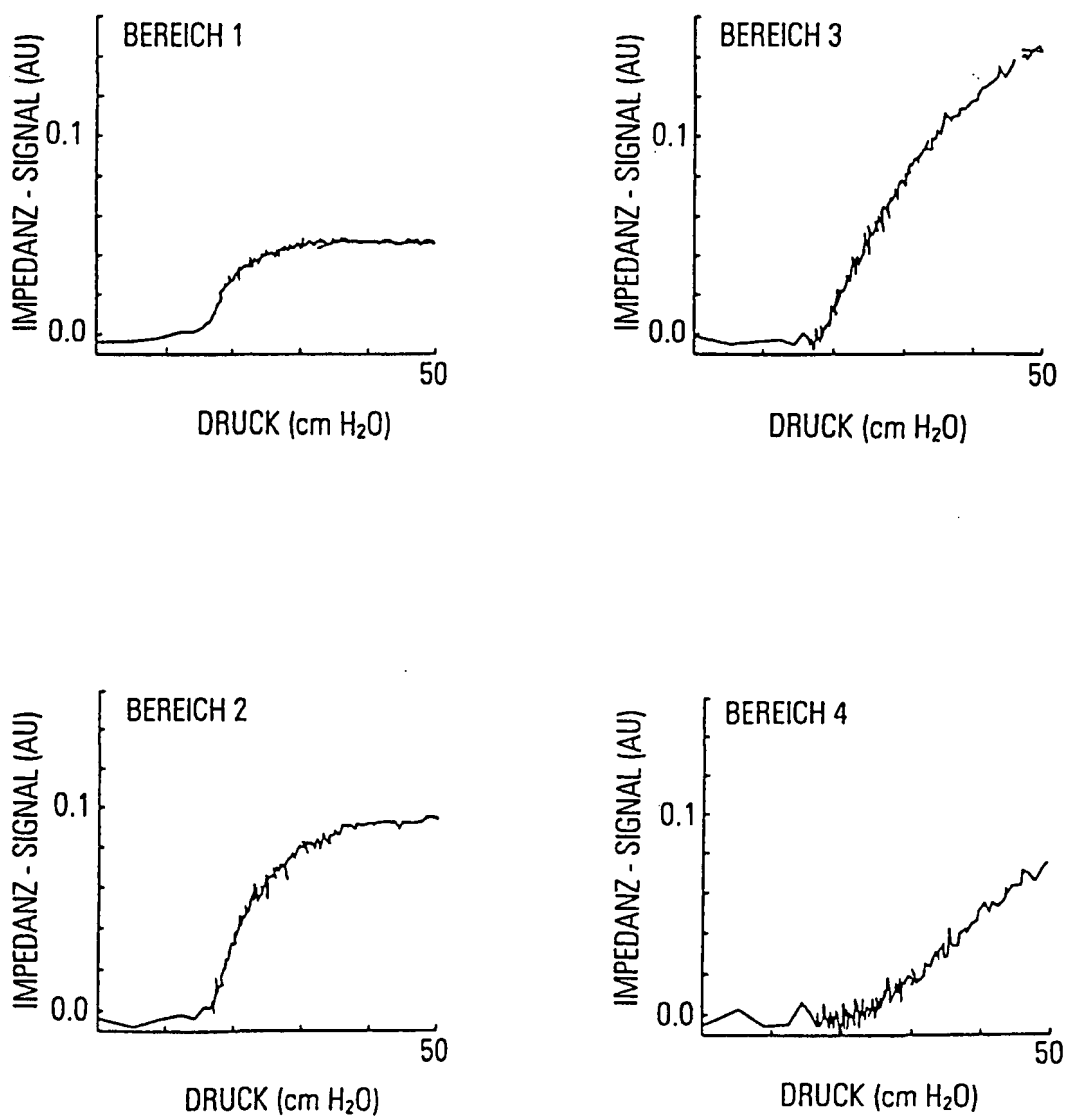
5 a control unit which is connected to the artificial respiration unit and the processing unit, whereby the first respiration pressure value and the second respiration pressure value is fed from the processing unit to the control unit to control the artificial respiration.

10 28. Apparatus according to claim 27, wherein the lower respiration pressure of artificial respiration is controlled such that a predetermined pressure is artificially maintained in the lung, which just makes possible keeping open all the alveoli.

15 29. Apparatus according to claim 25 or 26, further comprising:

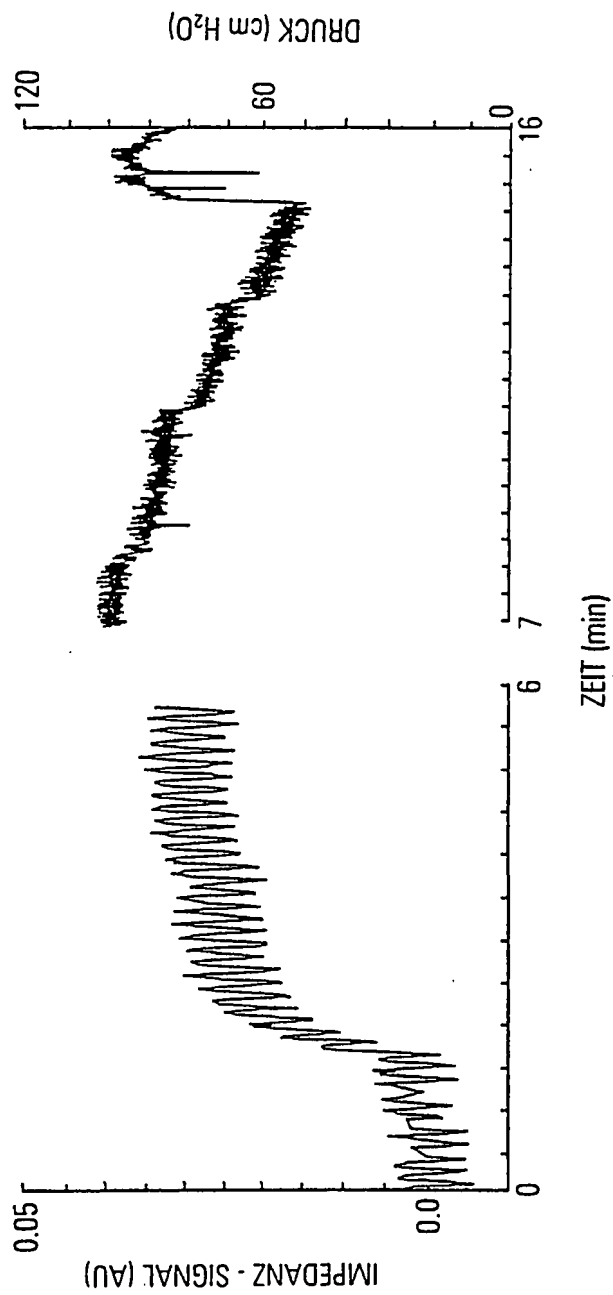
20 a monitoring unit for monitoring the first respiration pressure value and the second respiration pressure value.

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FIG.1

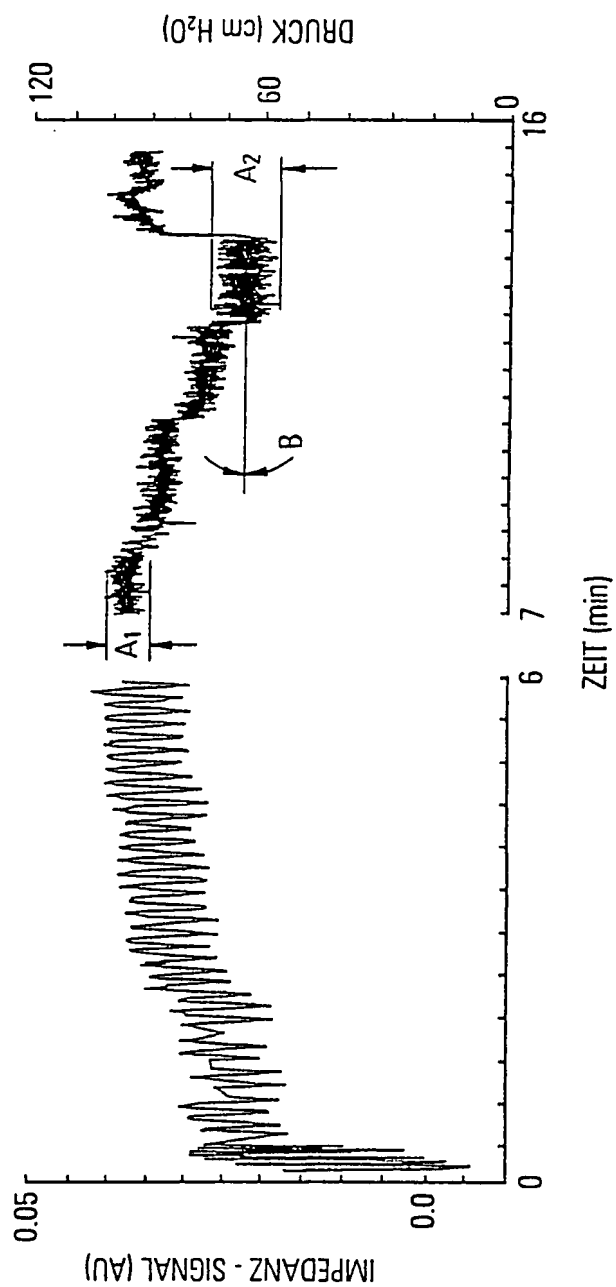
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FIG.2a

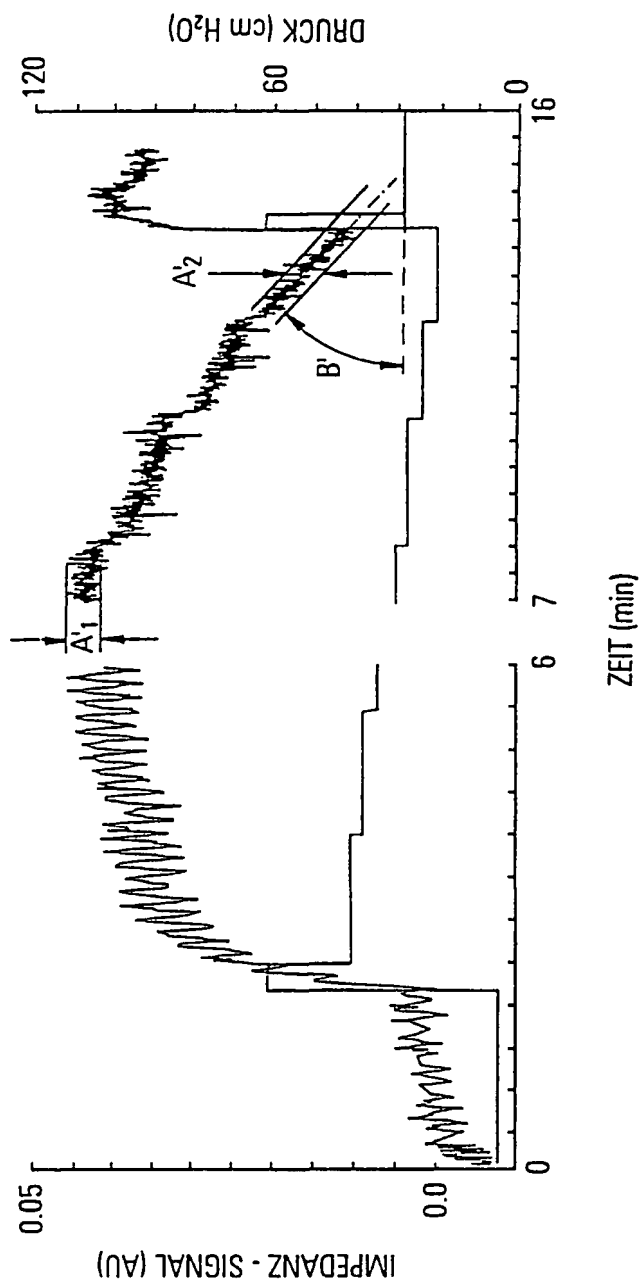


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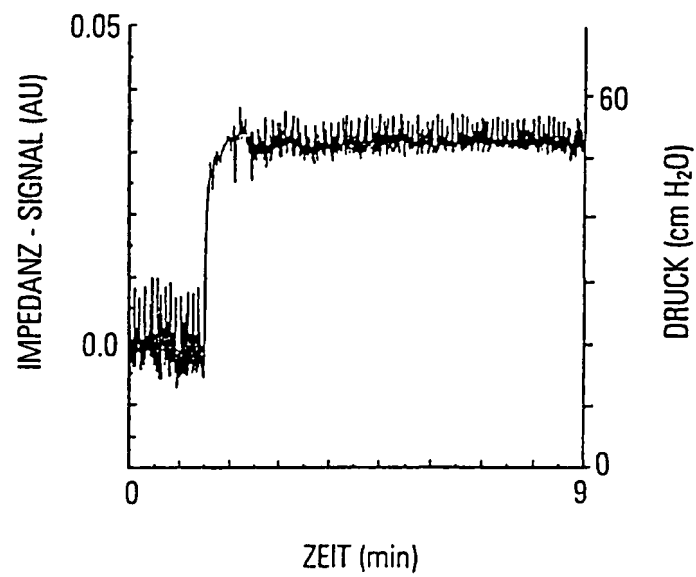
FIG. 2b



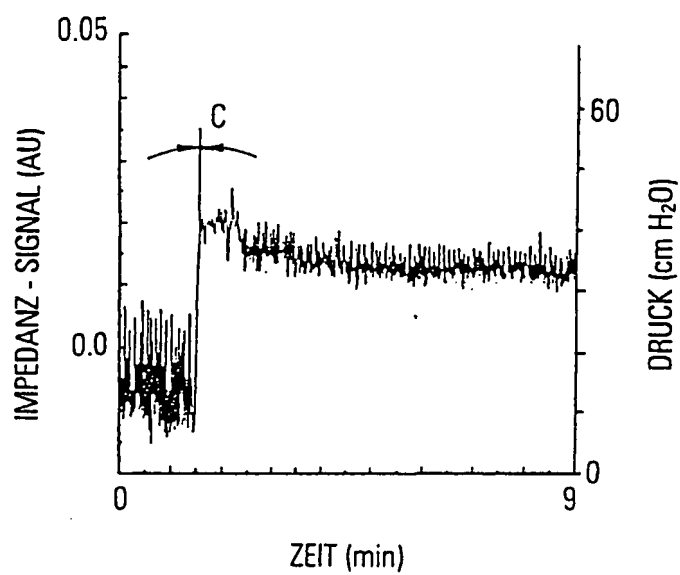
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FIG.2c

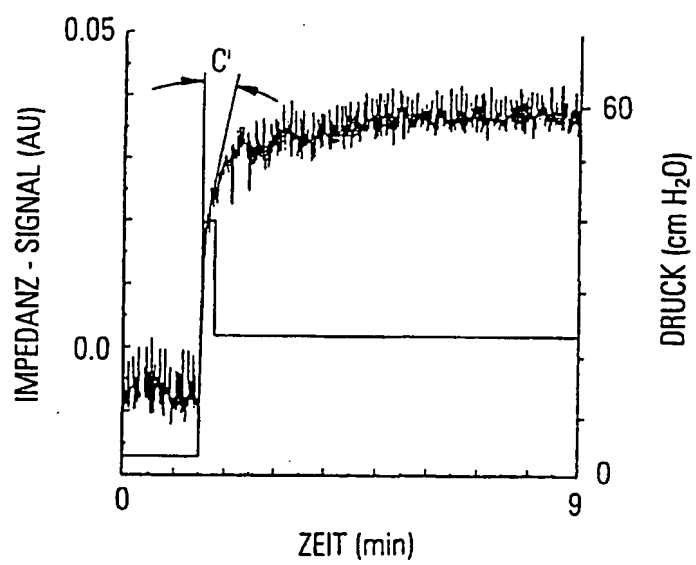
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FIG.3a

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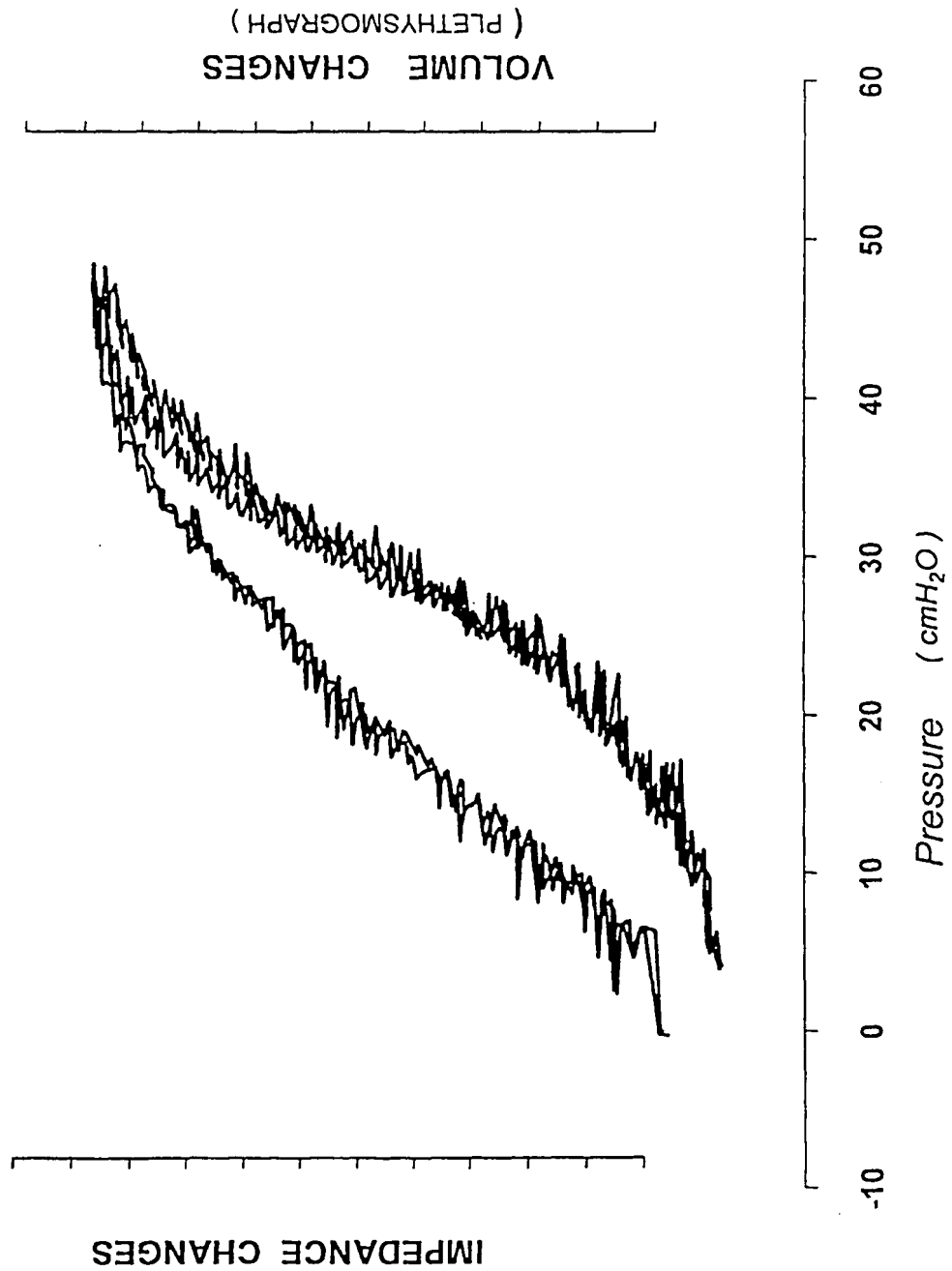
FIG.3b

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FIG.3c

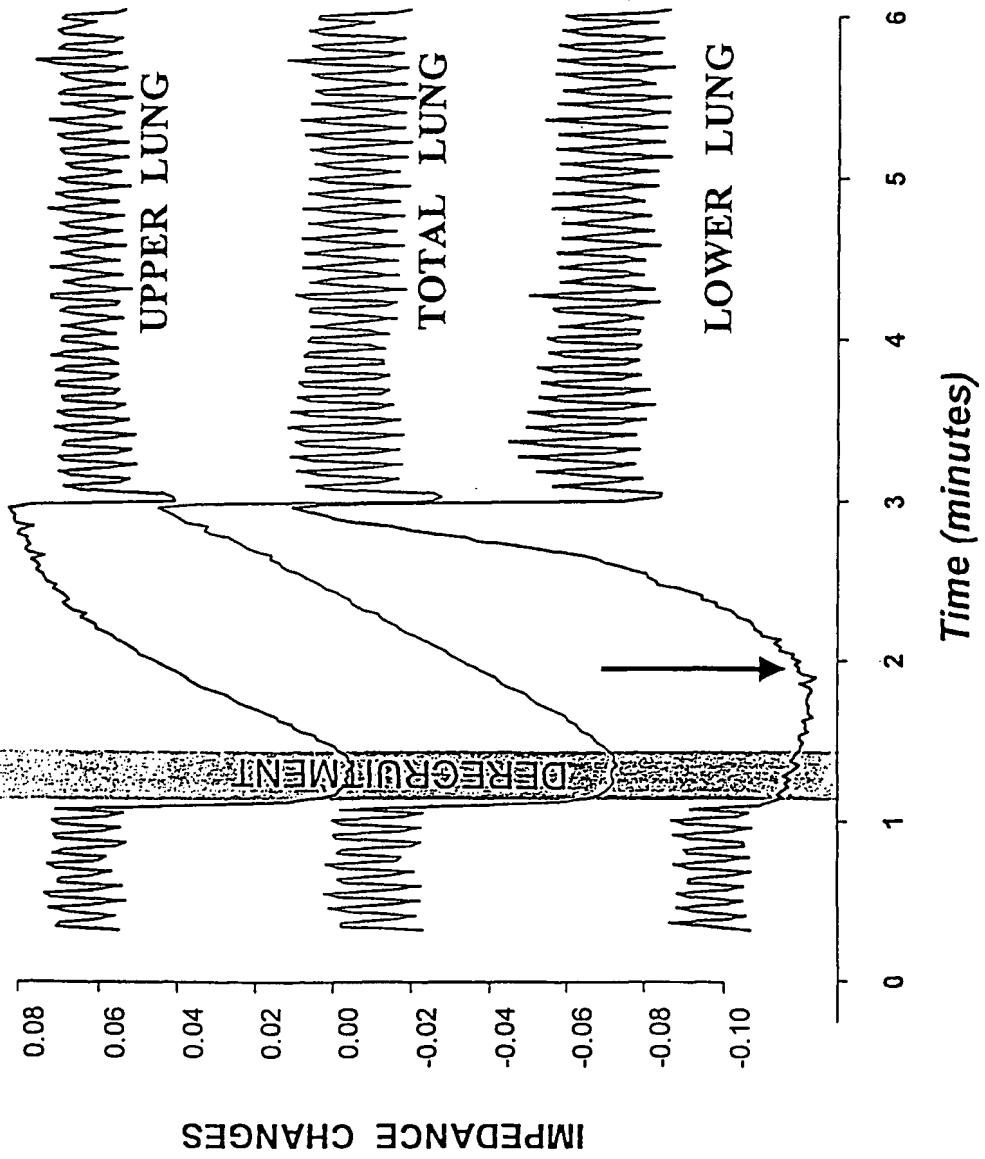
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FIG.4

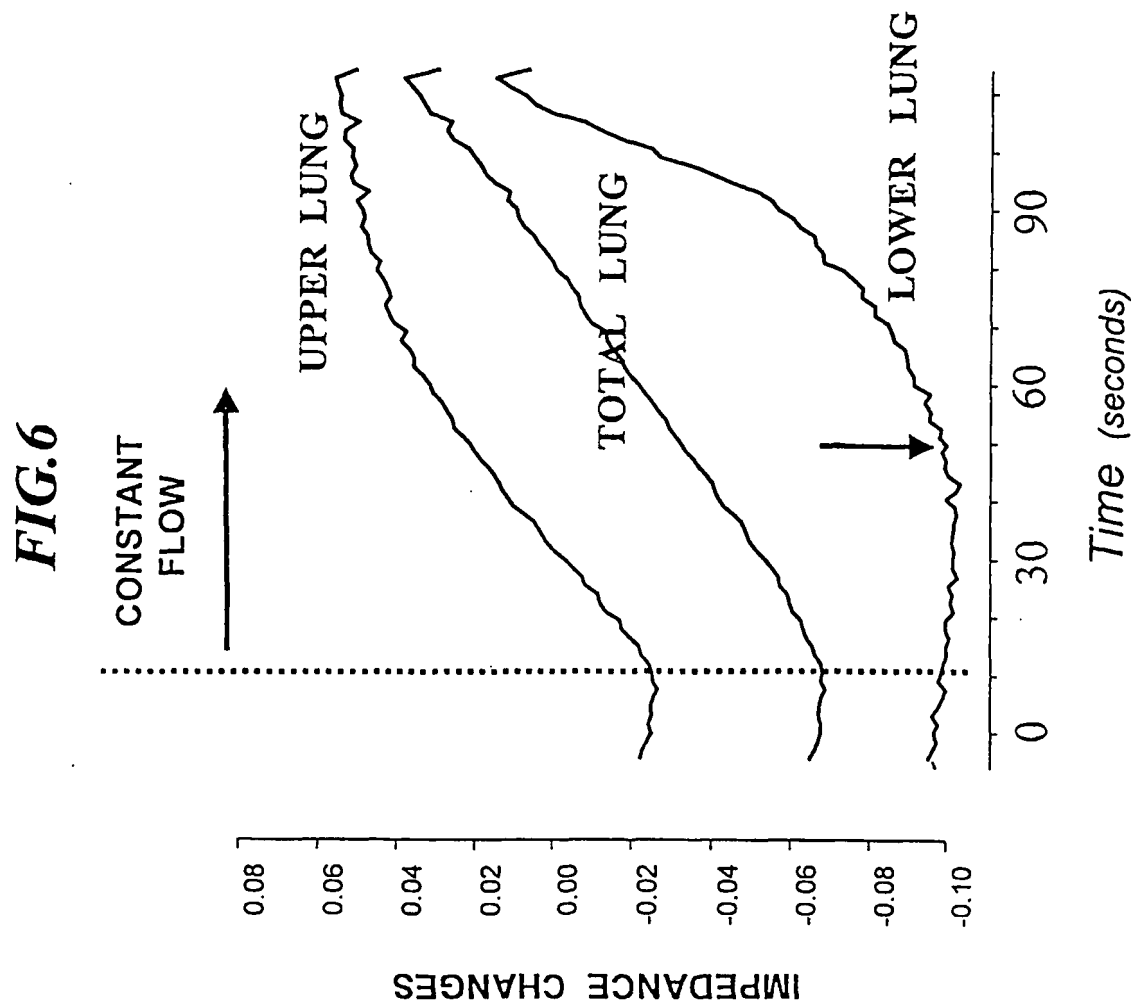


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FIG.5

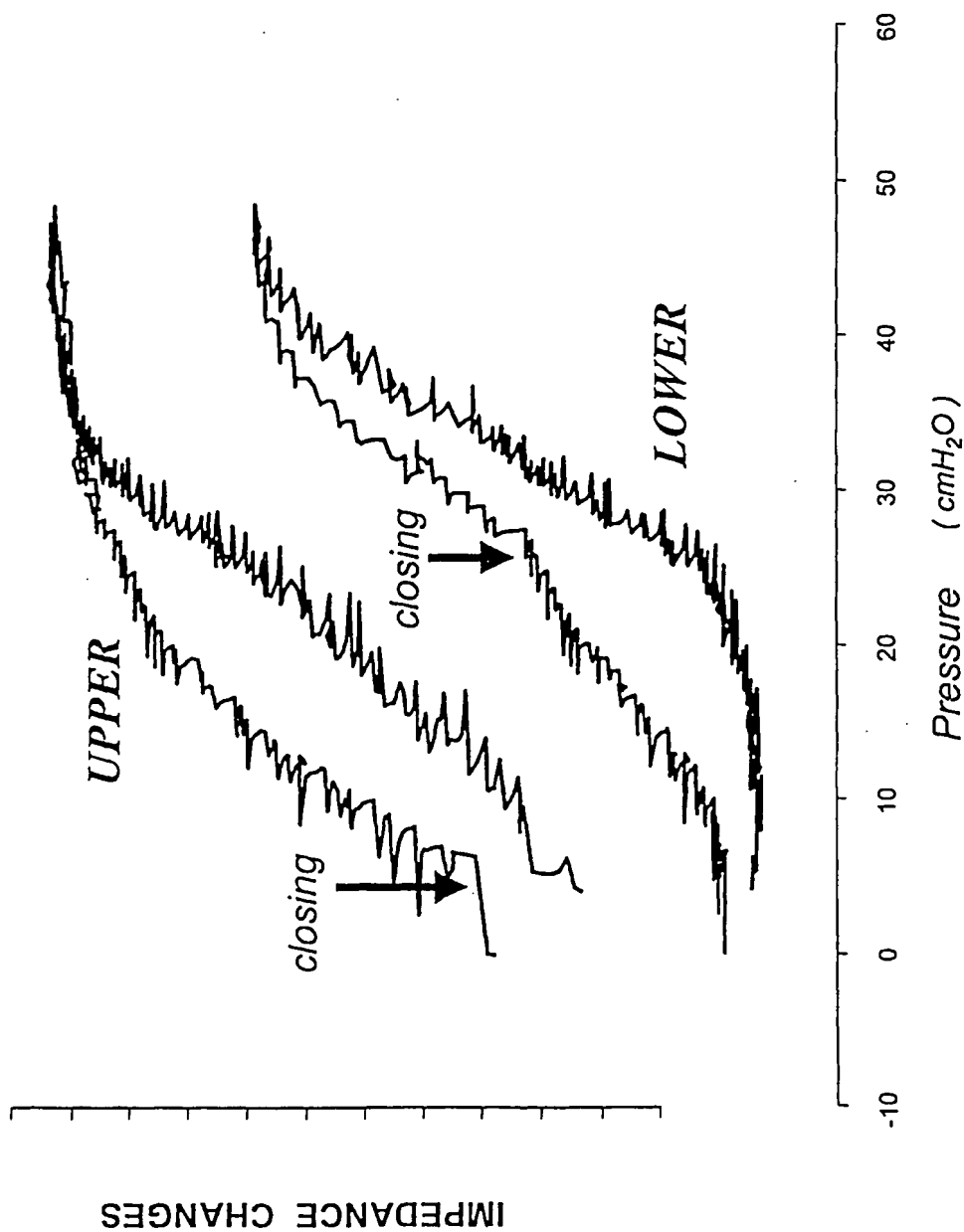


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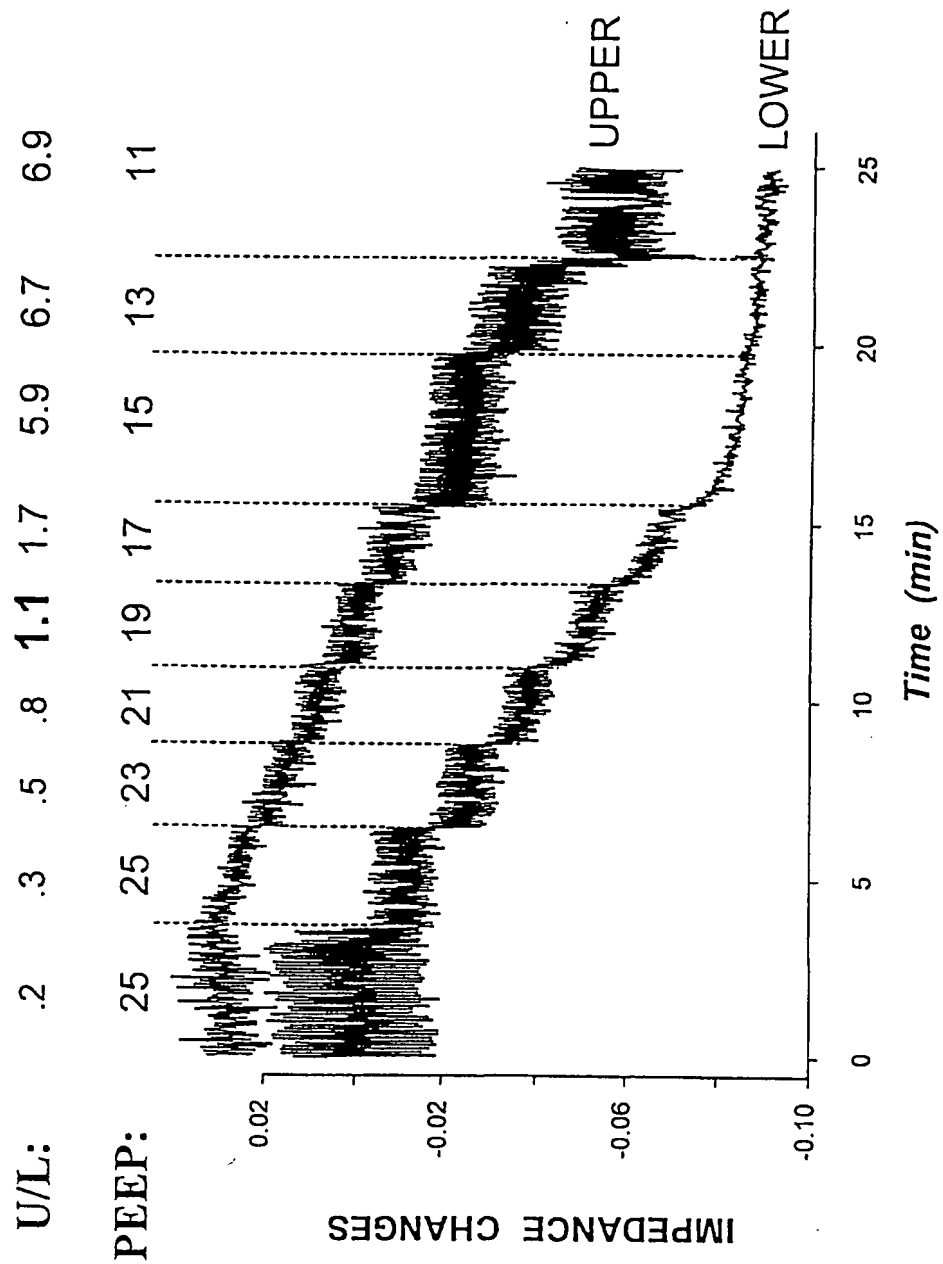
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FIG. 7

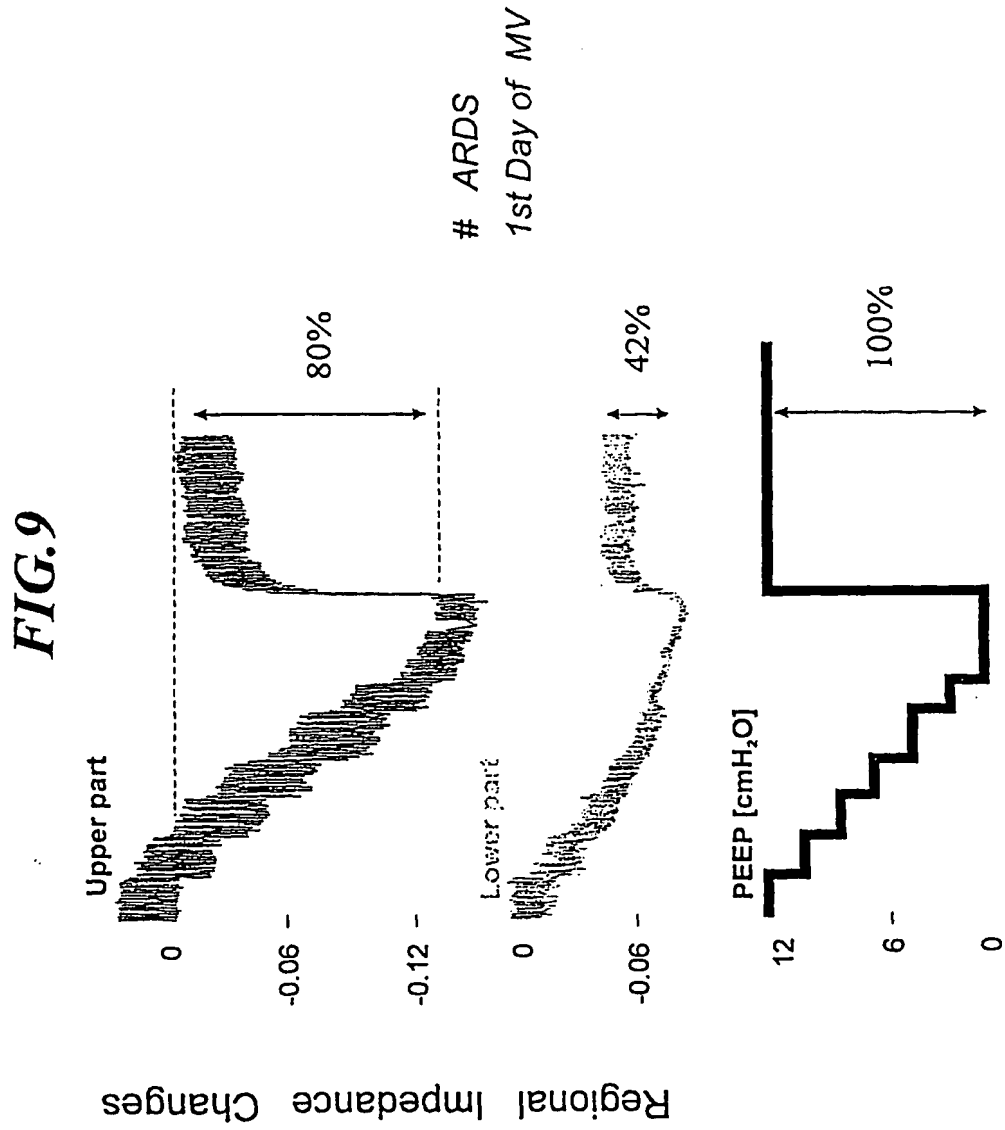


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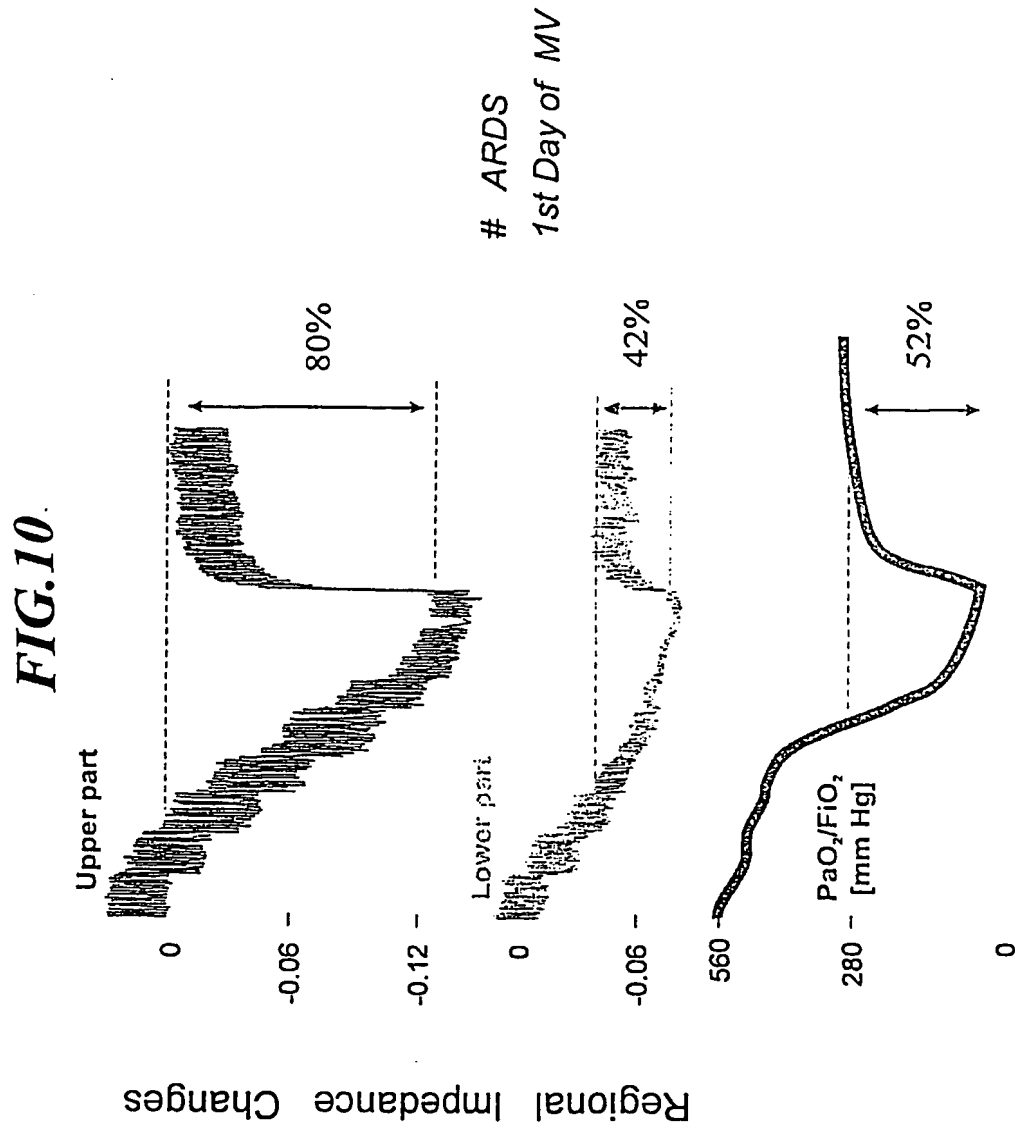
FIG.8



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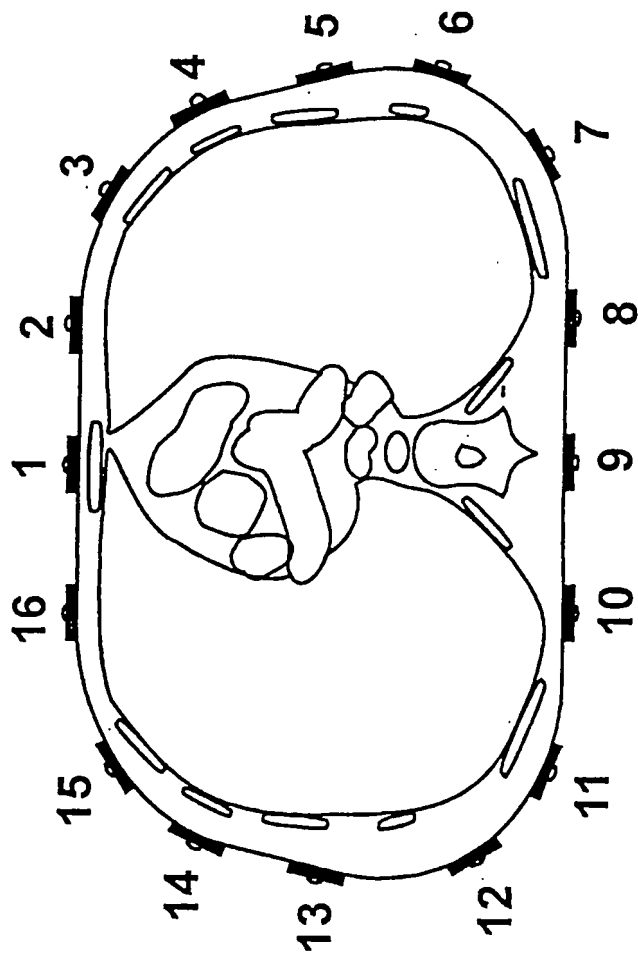


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FIG.11



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FIG.12

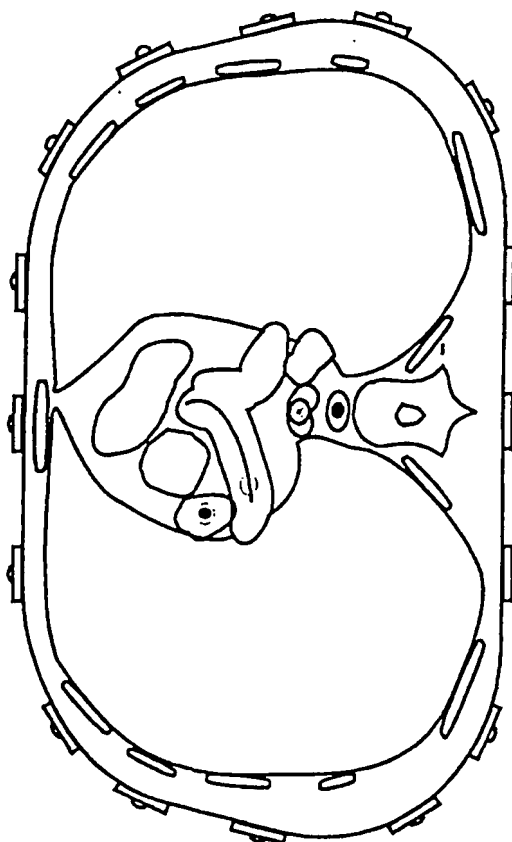
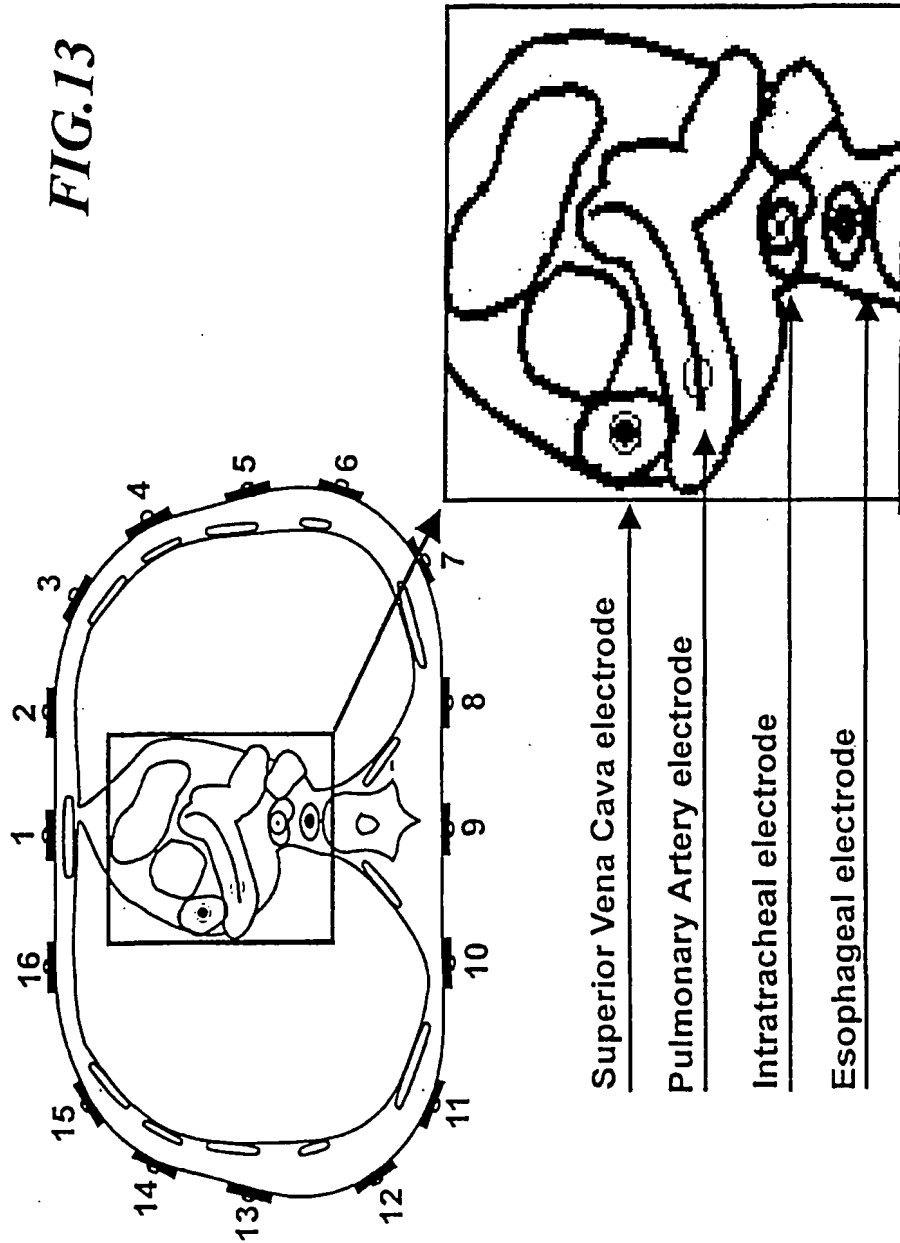


FIG.13



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FIG.14

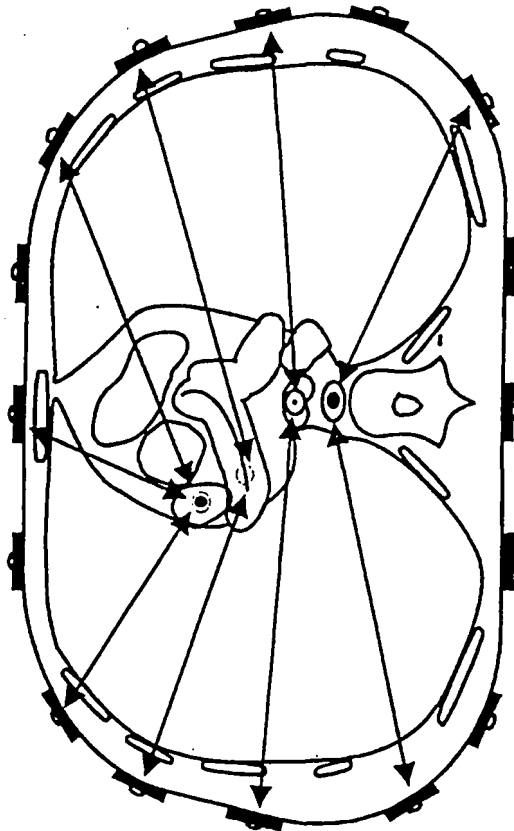


FIG.15

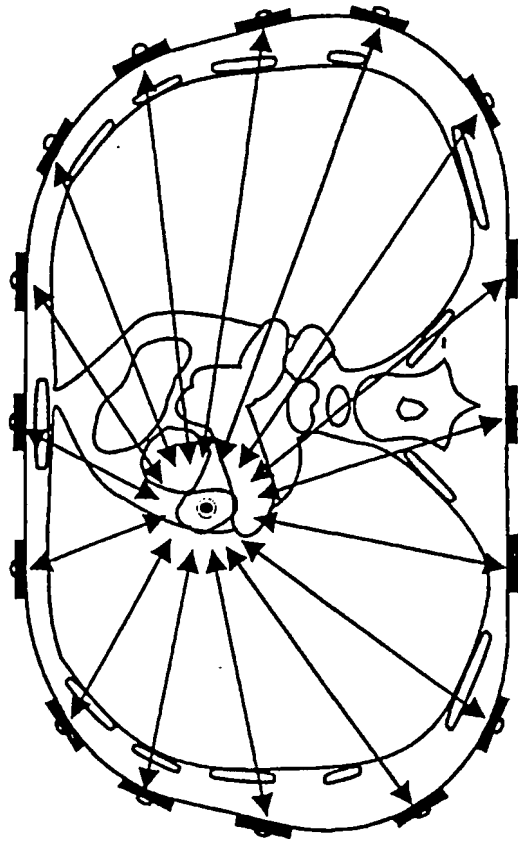
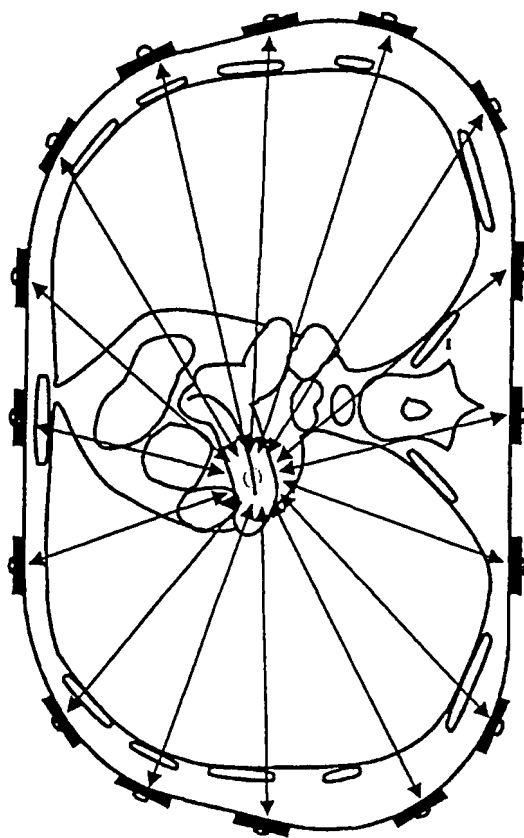
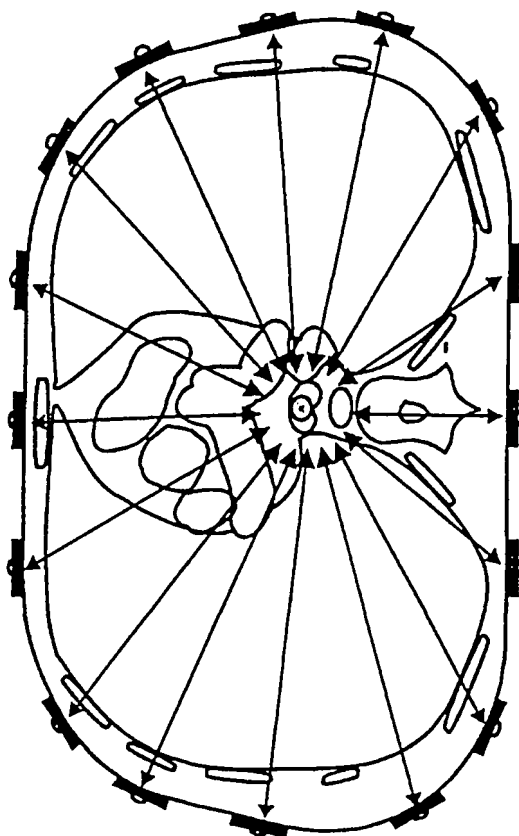


FIG.16

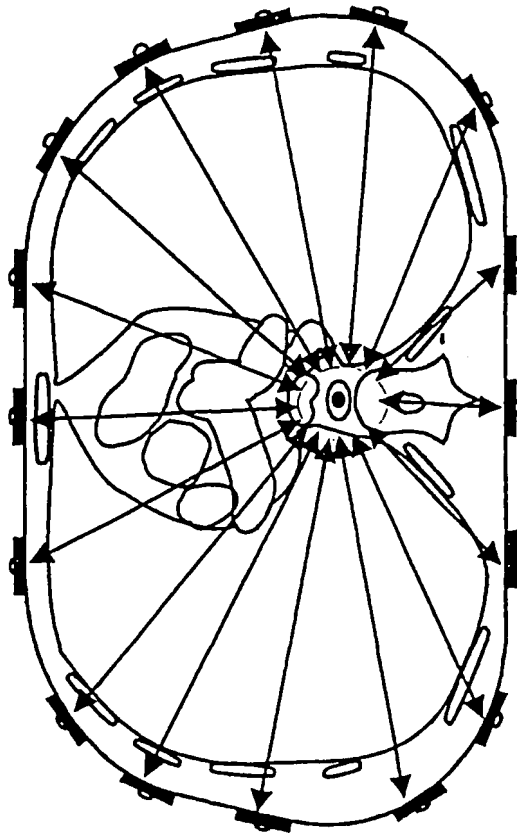


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FIG.17

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FIG.18



INTERNATIONAL SEARCH REPORT

Int'l Application No

PCT/EP 99/09699

A. CLASSIFICATION OF SUBJECT MATTER

IPC 7 A61B5/05 A61B5/08

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)

IPC 7 A61B A61M

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic data base consulted during the international search (name of data base and, where practical, search terms used)

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category *	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
A	DIJKSTRA A M ET AL: "REVIEW CLINICAL APPLICATIONS OF ELECTRICAL IMPEDANCE TOMOGRAPHY" JOURNAL OF MEDICAL ENGINEERING & TECHNOLOGY, GB, BASINGSTOKE, HANTS, no. 3, May 1993 (1993-05), pages 89-98, XP000700151 page 91, left-hand column, line 14 -page 92, right-hand column, line 23 -- -/--	1,25

☒ Further documents are listed in the continuation of box C.

☒ Patent family members are listed in annex.

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Date of the actual completion of the international search

19 April 2000

Date of mailing of the international search report

02/05/2000

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INTERNATIONAL SEARCH REPORT

Int. Application No

PCT/EP 99/09699

C.(Continuation) DOCUMENTS CONSIDERED TO BE RELEVANT

Category *	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
A	EUNG JE WOO ET AL: "MEASURING LUNG RESISTIVITY USING ELECTRICAL IMPEDANCE TOMOGRAPHY" IEEE TRANSACTIONS ON BIOMEDICAL ENGINEERING, US, IEEE INC. NEW YORK, vol. 39, no. 7, 1 July 1992 (1992-07-01), pages 756-760, XP000294413 ISSN: 0018-9294 abstract page 757, left-hand column, line 48 -page 759, left-hand column, line 47 figures 1-7	1,25
A	EP 0 818 177 A (SIEMENS-ELEMA AB) 14 January 1998 (1998-01-14) the whole document	1,27-29
A	EP 0 745 402 A (LACHMANN) 4 December 1996 (1996-12-04) column 4, line 7 -column 8, line 10	1,27-29

INTERNATIONAL SEARCH REPORT

Information on patent family members

International Application No

PCT/EP 99/09699

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EP 745402	A	04-12-1996	JP 9099083 A	15-04-1997
			US 5738090 A	14-04-1998

at 11/98

Optimierung der Beatmung beim akuten Lungenversagen durch Identifikation physiologischer Kenngrößen

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Burkhard Lachmann, Rotterdam



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Es wird ein neuartiges, automatisiertes Beatmungsverfahren für Patienten mit einem akuten Lungenversagen vorgestellt, das auf der Identifikation der für den jeweiligen Patienten optimalen Beatmungsdrücke basiert. Im Gegensatz zu den üblichen Beatmungsmethoden wird bei diesem Verfahren ein Signal aus dem Körper des Patienten (der Sauerstoffpartialdruck im Blut) kontinuierlich zurückgekoppelt, was erstmals eine regelmäßige Erfolgskontrolle der Beatmung ermöglicht.

Das Verfahren wurde auf einem Personal Computer unter LabVIEW® implementiert. Das dem Therapiekonzept zugrunde liegende medizinische Expertenwissen wurde in Form von „wenn ... dann“ – Regeln verbalisiert und als Fuzzy-Expertensystem in die Ablaufsteuerung integriert.

Optimal Artificial Ventilation by Identification of Physiological Parameters

A new automatic concept for artificial ventilation in patients with acute respiratory distress syndrome (ARDS) is presented. The concept features feedback of blood oxygen pressure in order to identify the optimal ventilation pressures and to monitor therapy progress.

The concept has been implemented on a PC under LabVIEW®. To represent the medical expert knowledge in a convenient way within the computer, a Fuzzy expert system has been developed and integrated into the software.

1 Einleitung

Auf den Intensivstationen in der Bundesrepublik Deutschland werden pro Tag etwa 8000–10000 Patienten beatmet. Die am Beatmungsgerät einzustellenden Größen, z.B. der Beatmungsdruck und die Atemfrequenz, werden meistens nach bewährten Standards ausgewählt, dann aber konstant gelassen und zu selten an die sich wandelnden Bedürfnisse der Patienten angepasst.

Zur Beurteilung des Beatmungserfolges wird in der Regel die arterielle Blutgasanalyse verwendet, bei der die Partialdrücke von Sauerstoff und Kohlendioxid (paO_2 und $paCO_2$) im Blut bestimmt werden. Allerdings werden diese Werte oft nur 1–4 mal pro Tag bestimmt. Wenn man bedenkt, daß ein Mensch pro Tag mehr als 20000 Atemzüge ausführt, wird klar, daß diese „Abtastfrequenz“ für eine wirkliche Beurteilung des Gasaustausches gerade bei kritischen und instabilen Patienten viel zu gering ist.

Zu dieser Gruppe gehören Patienten mit einem akuten Lungenversagen (ARDS = *acute respiratory distress syndrome*), einer Krankheit, die auch heute noch eine Sterblichkeit von 50 % besitzt [1]. Aufgrund eines Mangels an oberflächenaktiven Substanzen (sogenannter *surfactant*) kollabieren bei diesen Patienten große Teile der Lunge (sogenannte *Atelektase*) und stehen daher für den Gasaustausch nicht mehr zur Verfügung.

Ein erklärtes Therapieziel muß es sein, diese kollabierten Lungenareale möglichst frühzeitig und gleichzeitig schonend wieder zu „öffnen“, um Folgeerkrankungen und konsekutives Organversagen zu vermeiden. Die Wahl der Beatmungsdrücke hat hierauf einen großen Einfluß. Wie kürzlich von Amato et al. gezeigt wurde [2], läßt sich bereits durch eine vergleichsweise einfache Strategie zur Wahl der Drücke die Lungenfunktion von ARDS-Patienten deutlich verbessern und die Mortalität halbieren.

Im folgenden wird nun ein Verfahren vorgestellt, das ein noch größeres Potential zur Reduktion der Sterblichkeit erwarten läßt. Bei dem sogenannten „open lung concept“ wird die Lunge durch kurzfristige Überblähung nicht nur geöffnet, sondern durch Identifikation des Kollapspunktes und entsprechende Wahl der Beatmungsdrücke auch offen gehalten [3].

Die manuelle Ermittlung der individuell verschiedenen Öffnungs- und Schließdrücke ist allerdings mühsam und zeitaufwendig. Um das Konzept auch für den klinischen Alltag praktikabel zu machen, empfiehlt es sich, diese Beatmungsstrategie mit Hilfe eines Computers zu automatisieren.

2 Formen der künstlichen Beatmung

Die Hauptaufgabe der Lunge ist der Gasaustausch, d.h. die Versorgung des Körpers mit Sauerstoff und der Abtransport von Kohlendioxid. Für den Fall, daß ein Mensch den Gasaustausch nicht mehr selbständig bewerkstelligen kann, muß er künstlich beatmet werden.

Genau wie bei der Spontanatmung muß während der künstlichen Beatmung Frischluft aus der Umgebung in die Lungenbläschen gebracht werden. Bei diesen sogenannten „Alveolen“ handelt es sich um sackförmige Ausstülpungen (Bild 1) mit einem mittleren Durchmesser von ca. 70 µm, über deren Gesamtoberfläche der eigentliche Gasaustausch stattfindet.

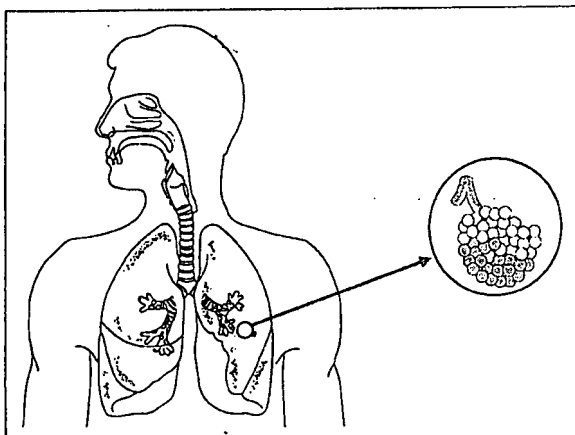


Bild 1: Bronchialbaum und Alveolen (ein gesunder Erwachsener besitzt ca. 300 Mio. Alveolen mit einer Gasaustauschfläche von ca. 100 m²).

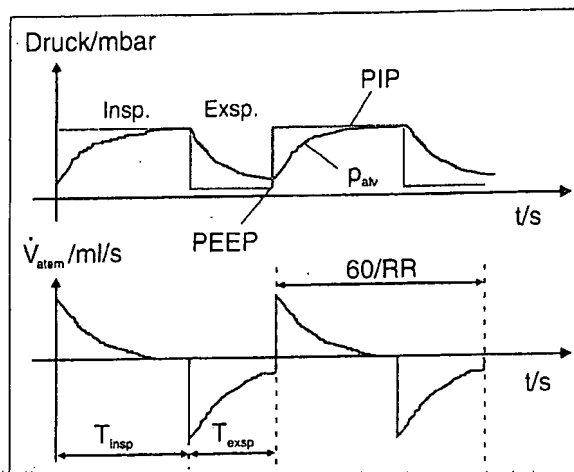


Bild 2: Druckkontrollierte Beatmung.

Beim *spontanatmenden* Menschen wird in den Alveolen durch Anspannung der Atemmuskulatur ein Unterdruck erzeugt und dadurch Luft angesaugt. Bei der heute üblichen Form der *künstlichen* Beatmung wird hingegen am Mund des Patienten ein Überdruck appliziert, durch den Luft in die Lunge gedrückt wird (sogenannte „Überdruckbeatmung“).

Generell wird zwischen *unterstützender* und *kontrollierter* Beatmung unterschieden [4]. Bei der unterstützenden (assistierten) Beatmung wird die Atemaktivität des Patienten registriert. Diese Form der Beatmung wird z.B. in der Schlußphase einer Beatmung kurz vor dem Übergang zur Spontanatmung eingesetzt. Hingegen wird die kontrollierte Beatmung eingesetzt, wenn eine vollständige Kontrolle über die Atmung des Patienten erwünscht oder nötig ist. Bei der kontrollierten Beatmung unterscheidet man zwei Formen, die *druckkontrollierte* und die *volumenkontrollierte* Beatmung.

2.1 Druckkontrollierte Beatmung

Bei diesem Beatmungsmodus wird der Beatmungsdruck sowohl während der Einatemungsphase (Inspiration) als auch während der Ausatemungsphase (Expiration) vorgegeben. Die zugehörigen Drücke nennt man *PIP* (*peak inspiratory pressure*) und *PEEP* (*positive endexpiratory pressure*). Der Alveolardruck p_{alv} pendelt entsprechend zwischen diesen beiden Druckwerten, Bild 2.

Weitere frei wählbare Größen sind die Beatmungsfrequenz *RR* (*respiratory rate*) und das Ein- zu Ausatemungsverhältnis ($I/E = \text{inspiration to expiration ratio}$). Hierbei gilt

$$RR = \frac{1}{T_{insp} + T_{exp}} \cdot \frac{60}{\text{min}} \quad (1)$$

und

$$I/E = \frac{T_{insp}}{T_{exp}} \quad (2)$$

mit T_{insp} der Inspirationsdauer und T_{exp} der Expirationsdauer. Das während eines Atemzuges bewegte

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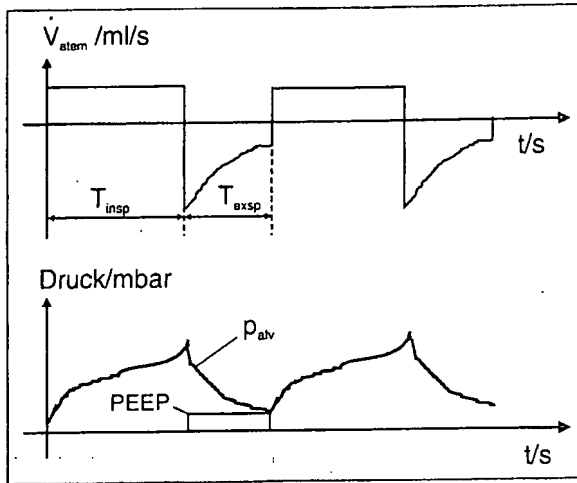


Bild 3: Volumenkontrollierte Beatmung.

Luftvolumen wird Tidalvolumen V_T genannt. Unter der Voraussetzung eines Leckage-freien Systems und des stationären Falls gilt

$$V_T = \int_0^{t+T_{\text{insp}}} \dot{V}_{\text{aterm}} dt = \int_0^{t+T_{\text{exp}}} \dot{V}_{\text{aterm}} dt. \quad (3)$$

2.2 Volumenkontrollierte Beatmung

Bei dieser Beatmungsform wird dem Patienten während der Inspiration ein konstanter Atemstrom aufgeprägt. Wie auch bei der druckkontrollierten Beatmung erfolgt die Expiration passiv auf einen vorgewählten PEEP-Wert. Bild 3 zeigt eine Skizze der entsprechenden Signalverläufe.

Im Gegensatz zu einer druckkontrollierten Beatmung kann bei einer volumenkontrollierten Beatmung ein bestimmtes (einstellbares) Tidalvolumen garantiert werden, weshalb diese Beatmungsform von manchen Intensivmedizinern bevorzugt wird. Natürlich gilt im stationären Fall wiederum Gl. (3). Der Nachteil dieses Beatmungsmodus ist jedoch, daß die Amplitude von p_{alv} nicht kontrolliert wird. Bei Patienten mit einer steifen Lunge können so unter Umständen Druckspitzen entstehen, die das Lungengewebe durch Überdehnung zum Reißen bringen können (sogenanntes „Barotrauma“). Ferner kann es bei Lungen mit inhomogenen Gewebeeigenschaften zu unerwünschten lokalen Luftverschiebungen innerhalb der Lunge kommen (sogenannte „Pendelluft“). Im Rahmen des hier vorgestellten Projektes wurde daher der druckkontrollierte Beatmungsmodus verwendet.

3 Modellbildung

Zum besseren Verständnis eines Prozesses und zur Auslegung von Regelungen oder Steuerungen empfiehlt es sich generell, ein für die Applikation geeignetes Prozeßmodell zu entwickeln. Eine systemtheoretische Modellbildung der beatmeten Lunge sollte die folgenden drei Teilsysteme beinhalten: „Beatmungsgerät“, „Atemmechanik“ und „Gasaustausch“, Bild 4.

Die Eingangsgrößen dieses Prozesses sind neben den Beatmungsdrücken PIP und PEEP die Atemfrequenz RR, das Einatmungs- zu Ausatmungsverhältnis I/E und die Sauerstoffkonzentration f_{O_2} (20 ... 100 %). Die Ausgangsgrößen sind die im arteriellen Blut herrschenden Partialdrücke pa_{O_2} und pa_{CO_2} , während das aktuelle Füllungsvolumen V_{lunge} und der alveoläre Druck p_{alv} innere Zustände des Prozesses sind, über die bisher allerdings wegen fehlender meßtechnischer Voraussetzungen kaum Aussagen gemacht werden konnten.

Insbesondere die Teilsysteme „Atemmechanik“ und „Gasaustausch“ sind stark nichtlinear. Es empfiehlt sich daher, sowohl das Kleinsignal- wie auch das Großsignalverhalten zu analysieren.

3.1 Kleinsignalverhalten

Die Lunge wird über ein sich zunehmend verzweigendes Röhrensystem (den sogenannten „Bronchialbaum“) belüftet. In diesem aus bis zu 23 Generationen bestehenden Röhrensystem ist ein großer Teil des Strömungswiderstandes $R_{\text{ström}}$ (resistance) lokalisiert, insbesondere in den Generationen 3–6 [5; 6].

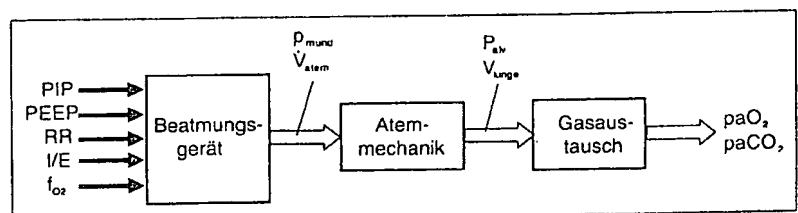
Der zweite atemmechanische Parameter der Lunge, die Volumendehnbarkeit C_{rs} , wird hingegen durch die elastischen Eigenschaften der peripheren Gewebestrukturen bestimmt. Sie wird auch als „compliance“ bezeichnet. Bild 5 zeigt ein elektrisches Ersatzschaltbild der Lungenmechanik.

Da der Munddruck eines spontanatmenden Menschen gleich dem Atmosphärendruck ist, gilt $p_{\text{mund}} = 0$. Aus dem Spannungsumlauf ergibt sich für die Lungenmechanik die folgende Differentialgleichung:

$$p_{\text{musk}}(t) + \frac{1}{C_{\text{rs}}} \cdot (V_{\text{lunge}}(t) - V_{\text{lunge},0}) + R_{\text{ström}} \cdot \dot{V}_{\text{aterm}}(t) = p_{\text{mund}} = 0 \quad (4)$$

Hierbei ist p_{musk} der durch die Atemmuskulatur während der Inspiration hervorgerufene (Unter-)Druck und $V_{\text{lunge},0}$ das in der Lunge befindliche Ruhe-Volu-

Bild 4: Blockschaltbild und Signalflüsse bei der Beatmung.



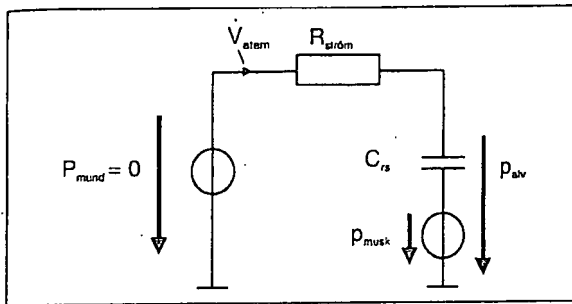


Bild 5: Ersatzschaltbild der Lungenmechanik beim spontanatmen den Menschen.

men. Die Dynamik dieses Systems wird durch die respiratorische Zeitkonstante

$$T_{rs} = R_{ström} \cdot C_{rs} \quad (5)$$

beschrieben. Für die Resistance und die Compliance gelten allgemein die folgenden Definitionsgleichungen:

$$R_{ström} = \frac{P_{mund} - P_{alv}}{\dot{V}_{atem}} \quad (6)$$

und

$$C_{rs} = \frac{V_{lunge} - V_{lunge,0}}{P_{alv} - P_{musk}} \quad (7)$$

Beim gesunden Erwachsenen liegen typische Werte bei $R_{ström} = 2 \dots 4$ mbar s/l und $C_{rs} = 230 \dots 290$ ml/mbar. Beide Parameter ändern sich jedoch stark mit dem Füllungszustand der Lunge und sind somit eine Funktion des Arbeitspunktes.

Beim beatmeten Patienten liegen andere Druckverhältnisse vor. Aufgrund der medikamentösen Muskel-Relaxation ist generell $p_{musk} = 0$, dafür wird nun ein von 0 verschiedener Munddruck appliziert. Durch den in der Atemröhre liegenden Schlauch („Tubus“) entsteht ein zusätzlicher Strömungswiderstand, der in der Regel turbulent ist und durch einen quadratischen Term berücksichtigt werden kann. Daher ändert sich Gl. (4) zu

$$P_{mund}(t) = \frac{1}{C_{rs}} \cdot (V_{lunge}(t) - V_{lunge,0}) + R_{ström} \cdot \dot{V}_{atem}(t) + R_{tubus} \cdot \dot{V}_{atem}(t) \cdot |\dot{V}_{atem}(t)| \quad (8)$$

vgl. [7]. Das zugehörige Ersatzschaltbild ist in Bild 6 dargestellt. Im Alveolus setzen sich die Partialdrücke der einzelnen Gasfraktionen nach dem Gesetz von Dalton zum Gesamtdruck p_{alv} gemäß

$$P_{alv} = P_{alv,O_2} + P_{alv,CO_2} + P_{alv,N_2} + P_{alv,H_2O} \quad (9)$$

zusammen. Hierbei entsteht der Wasserdampfpartialdruck p_{alv,H_2O} durch Anfeuchtung in den Atemwegen. Man erkennt, daß der alveoläre Sauerstoffpartialdruck selbst bei Beatmung mit 100%igem Sauerstoff (d.h., $P_{alv,N_2} = 0$ mmHg) in Ruhe um den Wasserdampfpartialdruck und den aktuellen Kohlendioxidpartialdruck P_{alv,CO_2} geringer ist als der Atmosphärendruck. Wegen

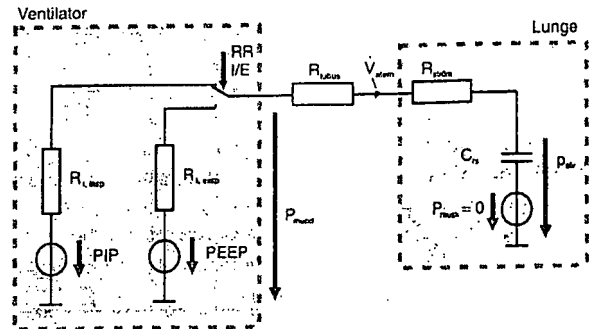


Bild 6: Elektrisches Ersatzschaltbild für die druckkontrollierte Beatmung. R_i bezeichnet den jeweiligen Innenwiderstand im Beatmungsgerät (modifiziert nach [8]).

$p_{alv,H_2O} = 47$ mmHg und $p_{alv,CO_2} = 39$ mmHg (jeweils bei 37 °C) gilt somit

$$P_{alv,O_2} \leq 674 \text{ mmHg} \quad (10)$$

Bei Normaldruckbeatmung kann dementsprechend auch der arterielle p_{aO_2} niemals über diesen Wert ansteigen.

Der Gasaustausch erfolgt gemäß dem 1. Fick'schen Diffusionsgesetz. So gilt z.B. für den Sauerstoffvolumenstrom aus den Alveolen ins Blut

$$\begin{aligned} \frac{dV_{O_2}}{dt} &= \frac{k \cdot A_{diff}}{l_{diff}} \cdot (p_{alv,O_2} - p_{aO_2}) \\ &= R_{diff} \cdot (p_{alv,O_2} - p_{aO_2}) \end{aligned} \quad (11)$$

Der diffundierende Volumenstrom ist daher neben dem Partialdruckgefälle direkt abhängig von der effektiven Gasaustauschfläche A_{diff} und umgekehrt proportional zur effektiven Diffusionsstrecke l_{diff} . Beim gesunden Menschen gleichen sich die Konzentrationsunterschiede zwischen den alveolären Partialdrücken und den im arteriellen Blut herrschenden Partialdrücken (p_{aO_2} und p_{aCO_2}) in kurzer Zeit ($\ll 1$ s) aus.¹

3.2 Großsignalverhalten

Das mechanische Großsignalverhalten einer Lunge läßt sich am besten durch ein pV-Diagramm beschreiben (Bild 7)². In diesem Diagramm läßt sich die Compliance C_{rs} auch durch

$$C_{rs} = \frac{V_T}{PIP - PEEP} \quad (12)$$

annähern (sogenannte „statische“ Compliance). Man erkennt, daß eine gesunde Lunge (Bild 7, links) über dem gesamten Druckbereich eine von Null verschiedene Compliance hat und bei typischen Beatmungsdrücken (z.B. $PIP = 20$ mbar, $PEEP = 5$ mbar) mit einem ausreichenden Tidalvolumen V_T versorgt werden kann.

Bei der schwerkranken Lunge (Bild 7, rechts) hingegen ist die Hysterese anders geformt und stärker ausge-

¹ Der Diffusionskoeffizient K ist für CO_2 etwa 20 mal größer als für O_2 .

² Man beachte, daß hier nicht das absolute Lungenfüllungsvolumen, sondern das auf $V_{lunge,0}$ bezogene relative Volumen dargestellt ist.

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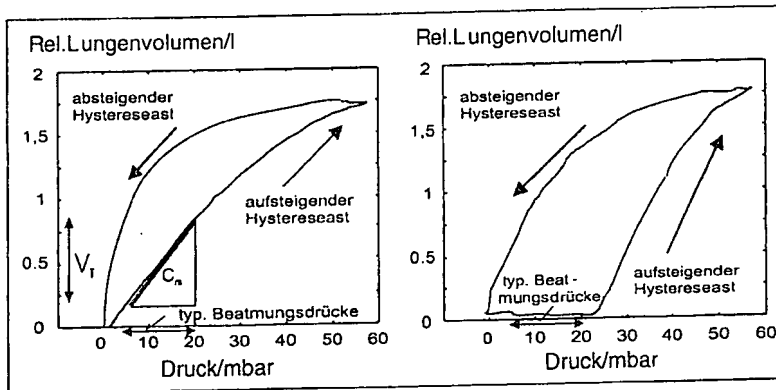


Bild 7: pV-Diagramme bei gesunder (links) und schwerkranker Lunge (rechts). Daten ermittelt am Tiermodell (Schwein, 33 kg).

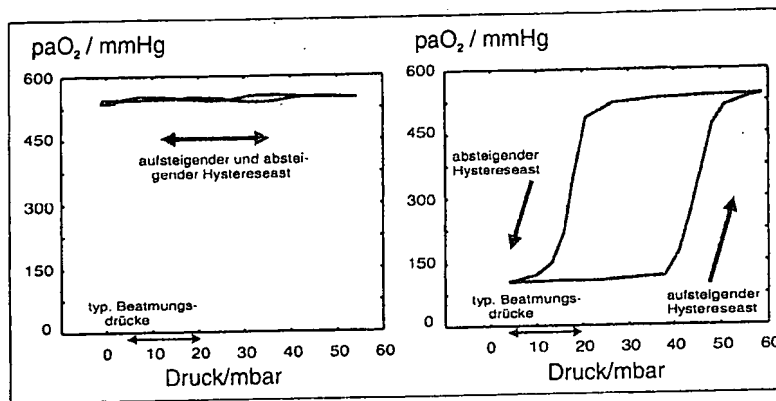


Bild 8: Arterieller Sauerstoffpartialdruck bei gesunder (links) und schwerkranker (rechts) Lunge. Beatmung mit 100 % Sauerstoff.

prägt. Insbesondere auf dem aufsteigenden Schenkel der Hysterese erreicht man bei typischen Beatmungsdrücken nur ein geringes Tidalvolumen. In diesem Bereich ist ein Großteil der Alveolen kollabiert und steht für den Gasaustausch nicht zur Verfügung. Noch deutlicher wird dies, wenn man das Großsignalverhalten des arteriellen Sauerstoffpartialdrucks betrachtet, vgl. Bild 8. Während es bei der gesunden Lunge (Bild 8, links) für paO_2 gar keine Hysterese gibt und die Wahl der Beatmungsdrücke unkritisch zu sein scheint, findet sich bei einer schwerkranken Lunge (Bild 8, rechts) eine noch ausgeprägtere Hysterese als im zugehörigen pV-Diagramm. Die Gasaustauschfläche A_{diff} ist hier in manchen Bereichen so stark reduziert, daß bei typischen Beatmungsdrücken nur bei hochprozentiger Sauerstoffgabe (90 ... 100 %) ein mit dem Leben vereinbarer paO_2 (> 80 mmHg) erreicht werden kann.

Eine solche Lunge läßt sich nur dann sinnvoll beatmen, wenn sie zunächst mit großen Drücken „geöffnet“ wird, um anschließend auf dem absteigenden Schenkel der Hysterese auch bei niedrigeren Drücken ein ausreichendes Ventilationsvolumen zu erzielen³.

Die auch bei der gesunden Lunge vorhandene, aber deutlich geringere Hysterese im pV-Diagramm (Bild 7, links) spiegelt die Tatsache wider, daß es auch physiologischerweise einen gewissen Anteil an kollabierten

Alveolen gibt, den man zwar druckgesteuert rekrutieren kann, der aber für das Erreichen des maximalen Sauerstoffpartialdrucks nicht notwendig ist.

4 Identifikation der Lunge

Für eine dem jeweiligen Patienten angepaßte Beatmung müssen das dynamische und das statische Verhalten der Lunge bekannt sein. Da sie sich unter Umständen innerhalb eines Tages deutlich verändern können, ist es wichtig, die Lunge jedes Patienten so oft wie nötig zu identifizieren.

4.1 Kleinsignalverhalten

Bei nicht zu großen Flüssen kann der durch den Tubus verursachte Widerstandsanteil als laminar betrachtet und $R_{ström}$ zugeschlagen werden. Der Strömungswiderstand und die Compliance lassen sich z.B. aus dem expiratorischen Teil der Atemflußkurve bestimmen. Betrachtet man $R_{ström}$ und C_{rs} während der Expiration als konstant, so gelten folgende Schätzgleichungen

$$\hat{R}_{ström} = \frac{PIP - PEEP}{\dot{V}_{atem,max}} \quad (13)$$

und

$$\hat{C}_{rs} = \frac{\hat{T}_{rs}}{\hat{R}_{ström}} \quad (14)$$

Hierbei wird die respiratorische Zeitkonstante \hat{T}_{rs} aus dem gemessenen expiratorischen Flußverlauf

³ Man beachte die Ähnlichkeit mit dem Hystereseverhalten von Magnetwerkstoffen. Auch bei einem Elektromagneten müssen zunächst alle Elementarmagnete durch eine hohe magnetische Erregung ausgerichtet werden, bevor man bei reduziertem elektrischem Strom eine ausreichende magnetische Flußdichte erreichen kann.

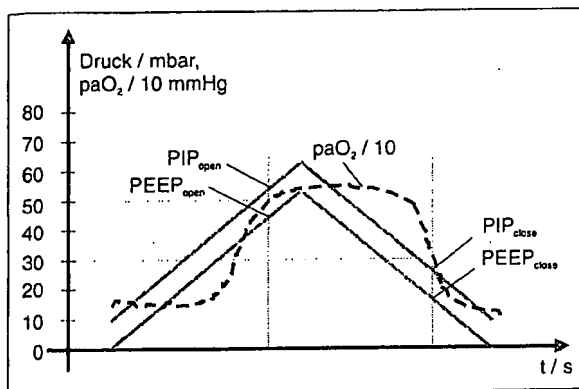


Bild 9: Druckverläufe zur Identifikation des Großsignal-Verhaltens einer schwerkranken Lunge.

nach der *Least Squares* (LS) Methode bestimmt [9; 10]. Wie sich gezeigt hat, ist der diagnostische Wert dieser beiden Parameter alleine nicht sehr hoch. Zwar ist es richtig, daß sie im Krankheitsfall spezifisch verändert sein können (z.B. ist $R_{ström}$ bei einer Bronchitis in der Regel erhöht), leider ändern sie sich aber auch mit dem Füllungszustand der Lunge, d.h. $R_{ström}$, $C_{rs} = f(V_{Lunge})$. Hinzu kommt, daß nach Bild 7 zumindest für C_{rs} auch eine druckabhängige Hysterese nachweisbar ist. Damit verändert sich C_{rs} auch noch als Funktion der zeitlichen Abfolge der Beatmungsdrücke. Durch diese Tatsache ist die isolierte Interpretation des Kleinsignalverhaltens am Arbeitspunkt nicht möglich. Für eine optimale Beatmung ist es daher wesentlich, auch das Großsignalverhalten der Lunge zu kennen.

4.2 Großsignalverhalten

Zur Identifikation eignet sich z.B. eine Druckrampe, mit der die Hysterese der Lunge einmal vollständig durchfahren werden kann. Bild 9 zeigt ein Beispiel (hier mit konstanter Druckdifferenz $PIP - PEEP$). Zur Bestimmung des Öffnungs- und Schließdrucks wurde definiert, daß eine kranke Lunge als „sicher offen“ anzusehen ist, wenn $paO_2 > 450$ mmHg ist (bei Beatmung mit 100 % Sauerstoff). In gleicher Weise gilt eine Lunge als „sicher geschlossen“ (zu einem großen Teil kollabiert), wenn $paO_2 < 300$ mmHg ist. Das Hystereseverhalten der Lunge bezüglich paO_2 ist durch die Öffnungs- und Schließdrücke vollständig bestimmt.

5 Optimale Beatmung von ARDS-Patienten

Das im folgenden vorgestellte Beatmungskonzept beruht auf den Vorarbeiten von Lachmann, Böhm und Mitarbeitern an der Universität von Rotterdam [3; 11; 12].

5.1 Das „open lung“ Konzept

In diesem neuartigen Beatmungskonzept wird die Identifikation des Großsignalverhaltens mit einer nachgeschalteten, erneuten Lungenöffnung und anschließender Dauerbeatmung bei möglichst niedrigen Drücken kombiniert. Als Feedback-Signal dient der kontinuierlich gemessene paO_2 , über den das Öffnen und der Kollaps der Lunge detektiert werden können. Bei der anschließenden Dauerbeatmung wird als „optimaler“ Arbeitspunkt die Beatmung mit

$$PEEP_{Dauerbeatmung} = PEEP_{close} + 2 \text{ mbar} \quad (15)$$

angesehen. Dadurch kann ein erneuter Kollaps der Lunge sicher vermieden werden. Bild 10 zeigt schematisch die Signalverläufe.

5.2 Automatisierung des „open lung“ Konzeptes

Die manuelle Bestimmung des jeweiligen Öffnungs- und Schließdrucks ist sehr zeitaufwendig und unter klinischen Bedingungen nur schwer praktikabel. Im Rahmen dieses Projektes wurde daher das „open lung“-Beatmungskonzept mit Hilfe eines Personal Computers und eines kontinuierlich arbeitenden Blutgassensors automatisiert, Bild 11. In der vorliegenden Version er-

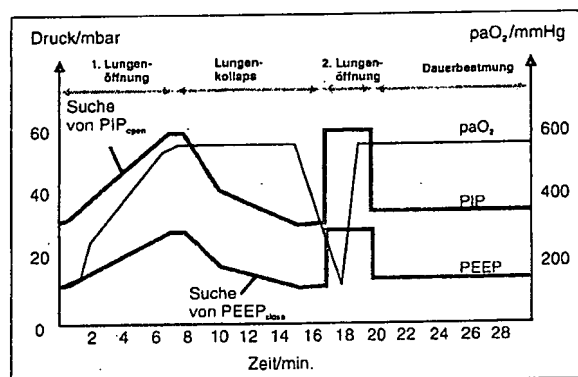


Bild 10: Neuartiges Konzept zur Identifikation und anschließenden Dauerbeatmung.

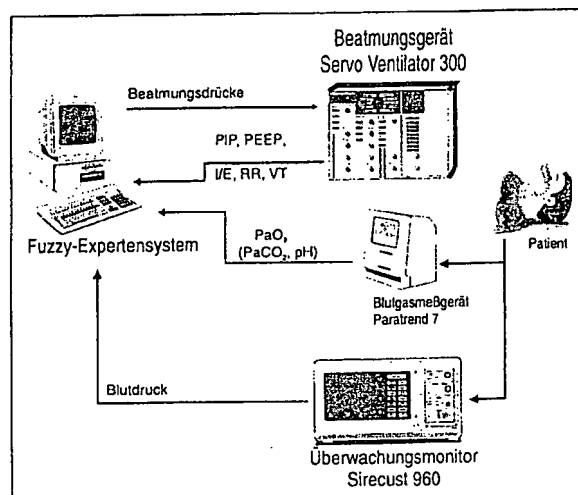


Bild 11: Anordnung der Hardware-Komponenten.

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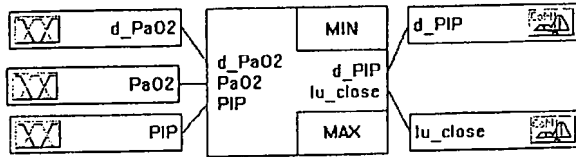


Bild 12: Ein Beispiel der implementierten Fuzzy-Regeln: die Änderung des inspiratorischen Drucks ist eine Funktion des Sauerstoffpartialdrucks, seiner Änderung und des aktuellen inspiratorischen Drucks.

folgt die Ablaufsteuerung der Beatmungsdrücke durch ein LabVIEW®-Programm, in das mit Hilfe einer Fuzzy-Toolbox das vorhandene medizinische Wissen in Form von insgesamt ca. 60 „wenn ... dann“-Regeln integriert wurde. Diese Regeln besitzen z.B. die Form

$$\begin{aligned} \text{wenn } paO_2 = \text{HOCH und } \Delta paO_2 = \text{KLEIN} \\ \text{und } PIP = \text{HOCH dann } \Delta PIP = \text{negativ} \end{aligned} \quad (16)$$

und bilden in ihrer Gesamtheit verschiedene mehrdimensionale, im allgemeinen nichtlineare Funktionen, siehe Bild 12. Die einzelnen Parameter der Regeln (z.B. „HOCH“, „KLEIN“) werden vom Arzt vorgegeben. Da sich ein bestimmter Druck bei einem schwerkranken Patienten anders auswirken kann als bei einem gesunden (postoperativ beatmeten) Patienten, können die Parameter der Regeln zusätzlich durch fuzzyfizierte Eingaben wie „Schweregrad der Lungenerkrankung“ oder „bekannter Bluthochdruck“ an die individuellen Bedürfnisse adaptiert werden.

Während eines Öffnungsvorgangs herrschen im Thorax relativ hohe Drücke, die das Herz und die Blutgefäße der Lunge kurzfristig komprimieren. Daher wurde zusätzlich eine Blutdrucküberwachung implementiert (Bild 11). Fällt der Blutdruck während einer Öffnungsprozedur zu stark, wird ein Alarm ausgelöst und der maximale Beatmungsdruck so lange reduziert, bis der Blutdruck wieder entsprechend gestiegen ist.

In der Dauerbeatmungsphase werden der Sauerstoffpartialdruck und der Munddruck kontinuierlich überwacht. Sinkt paO_2 ab, so besteht der Verdacht auf einen Lungenkollaps, z.B. weil sich das ARDS verschlechtert hat. Sinkt p_{mund} kurzfristig ab, so kann der Grund dafür in einer Diskonnektion liegen (z.B. weil der Patient durch das Pflegepersonal zur Schleimentfernung von der Beatmungsmaschine genommen und

nach dem Absaugen wieder konnektiert wurde). In beiden Fällen erfolgt eine erneute automatische Lungenöffnung.

5.3 Experimentelle Ergebnisse

Bild 13 zeigt ein Beispiel für die automatisierte Identifikation der paO_2 -Hysterese und die anschließende Weiterbeatmung im Tierversuch. Wie aus diesem Beispiel ersichtlich ist, kann eine ARDS-Lunge tatsächlich durch eine geeignete Beatmungsdruck-Trajektorie in ihrem Input-Output-Verhalten identifiziert und anschließend optimal therapiert werden. Gegenüber dem Anfangswert konnte der paO_2 durch die nur wenige Atemzüge dauernde Druck-Erhöpfung fast verdreifacht werden, obwohl PIP und $PEEP$ am Ende nur unwesentlich über den anfänglich eingestellten Werten lagen.

Der Verlauf des Blutdrucks während einer Öffnungsprozedur ist in Bild 14 gezeigt. Man erkennt einen klinisch tolerablen Blutdruckabfall, der mit den Beatmungsdrücken korreliert ist.

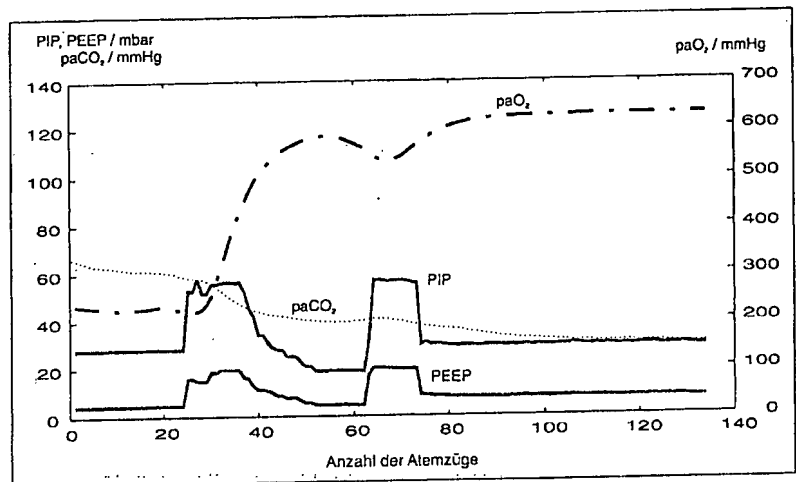


Bild 13: Öffnung einer schwerkranken Lunge (Schwein, 33 kg, ARDS).

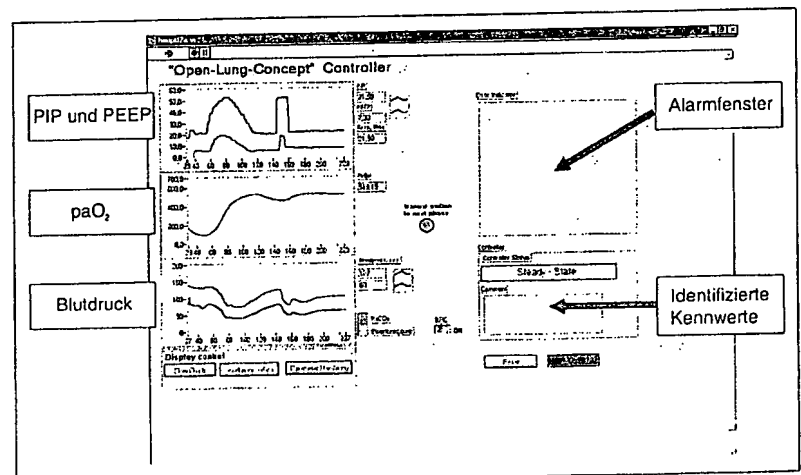


Bild 14: „User Interface in LabVIEW®. Die unterste Meßkurve zeigt den Verlauf des (systolischen und diastolischen) Blutdrucks während einer Öffnungs-Prozedur.

Bei der Entwicklung der Benutzer-Oberfläche wurde Wert auf eine übersichtliche und benutzerfreundliche Darstellung aller Informationen und Alarmer gelegt. Denn für den späteren klinischen Erfolg ist nicht nur die sichere Funktion, sondern auch die Ergonomie und die Transparenz entscheidend.

6 Zusammenfassung

Wie in vielen anderen Gebieten lassen sich auch in der Medizin durch den Einsatz von Computern und intelligenten automatisierten Verfahren große Verbesserungen erzielen und völlig neuartige Therapiekonzepte entwickeln, die ohne diese Hilfsmittel undenkbar wären.

Das gezeigte Beatmungsverfahren basiert auf der Identifikation des Übertragungsverhaltens einer Lunge. Lungenmechanik und Gasaustauschverhalten sind durch eine nichtlineare Dynamik und eine Hysterese gekennzeichnet. Insbesondere für ARDS-Lungen sind die arbeitspunktabhängigen Parameter $R_{ström}$ und C_{rs} von geringerer Aussagekraft als die Öffnungs- und Schließdrücke.

Es konnte gezeigt werden, daß sich eine ARDS-Lunge durch kurzfristige Druckerhöhung automatisiert öffnen und anschließend offen halten läßt. Der der Hysterese zugrunde liegende Surfactant-Mangel kann bei diesen Patienten durch eine geeignete Druck-Ablaufsteuerung kompensiert und ein fast normaler Gasaustausch gewährleistet werden.

Auch Patienten mit annähernd gesunden Lungen können von einer (einmaligen) Lungenöffnung und der Identifikation der für sie optimalen Beatmungsdrücke profitieren. Denn da auch bei einem lungengesunden Patienten gewisse Teile der Lunge kollabiert sind (Bild 7, links), kann es bei nicht erfolgter vollständiger Öffnung in den betroffenen Lungenarealen zu einem ständigen Wechselspiel zwischen Öffnung und Kollaps kommen. Die hierbei auftretenden Scherkräfte sind enorm hoch [7] und können ihrerseits die Lunge schädigen [13].

Ein Nachteil des vorgestellten Verfahrens ist die Verwendung des invasiven Blutgassensors. Die zugehörigen Sensorelemente sind Einmal-Artikel und mit DM 500,-/Stück teuer. Ein Zukunftsziel muß daher die Entwicklung eines blutgasunabhängigen Konzeptes sein.

Zum gegenwärtigen Zeitpunkt bietet das Beatmungskonzept zwei Gefahrenquellen: die mögliche

mechanische Überlastung des Lungengewebes (Gefahr eines Barotrauma) und die Möglichkeit des Blutdruckabfalls durch Kompression des Herzens. Speziell für das zweite Problem hat sich die Blutdruck-Überwachung bewährt, gegebenenfalls in Kombination mit einer Infusions-Gabe durch das ärztliche Personal.

Das automatisierte „open lung“ Beatmungskonzept wurde bisher in Tierversuchen ausgiebig und erfolgreich getestet. Die Erprobung in der Klinik steht kurz bevor.

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Is early kinetic positioning beneficial for pulmonary function in multiple trauma patients?

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Body positioning (kinetic therapy) is known to improve oxygenation in patients with impaired pulmonary function and ARDS. We have used body positioning prophylactically in trauma patients whose injury and pattern predispose to ARDS. This retrospective study reports the effects of early prophylactic (group P) versus late (group L) axial rotation on pulmonary function and the incidence of ARDS. Both groups were comparable in age, injury severity and the degree of thoracic injury. Systemic oxygenation was significantly better and the incidence of ARDS significantly lower in group P (group P: 34.3 per cent, group T: 74.1 per cent, $P < 0.05$). There was a tendency towards a lower incidence of pneumonia and a better survival in group P, which did not reach statistical significance. The duration of kinetic therapy and of ventilation was comparable in both groups. In this retrospective evaluation early prophylactic kinetic therapy was associated with a significantly lower incidence of ARDS compared with that instigated later. © 1998 Elsevier Science Ltd. All rights reserved.

positive effects on oxygenation⁵. However, in patients with associated bronchopleural fistula, which often develops in patients with thoracic injury, air is lost through the fistula⁶. The effects of medical therapy (surfactant replacement and nitric oxide application) have not been investigated sufficiently to reach conclusions about their efficacy and although promising results have been reported⁷, the high costs have to be considered.

Body positioning is an inexpensive *non invasive* method, which is known to improve oxygenation. The technique involves alternating prone and supine positioning or continuous axial rotation (kinetic therapy)^{8–10}. So far body positioning has only been applied for threatening ARDS, i.e. as a therapeutic measure when pulmonary function is deteriorating. It may be argued that this is rather late. A variety of pathogenetic changes occur before there is a clinically measurable fall in oxygen tension. These changes include an increase of pulmonary lymph flow for clearance of the rise of interstitial fluid. Deterioration of oxygenation represents an end point in a chain of events induced by microvascular injury. Initiation of therapy at this late stage may not be able to reverse the mechanisms causing the impairment of oxygenation.

It was our impression that there were a number of patients for whom body positioning was not able to fully reverse the impairment of oxygenation. In other patients pulmonary recovery occurred slowly and was associated with the risk of the development of further organ dysfunction. Therefore we began to test the kinetic device early after injury even without evidence of clinically measurable lung dysfunction. Patients known to be at risk of developing respiratory distress syndrome were submitted to early kinetic therapy as a prophylactic measure. With this regimen we have seen a number of patients with an uneventful clinical course following severe thoracic trauma or high severity of injury. The aim of this

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Introduction

Post-traumatic pulmonary insufficiency and adult respiratory distress syndrome (ARDS) continue to represent a major threat to the trauma patient. Not only is ARDS itself associated with a high mortality but it also predisposes to the development of multiple organ failure (MOF)^{1–3}. The treatment of lung failure is particularly difficult during the early post injury period.

Invasive methods of support, such as extracorporeal oxygenation, are considered inappropriate during the first week after injury because of the risk of severe bleeding⁴. No clinical studies have been reported on the use of other methods of artificial oxygenation (e.g. IVOX) for blunt trauma patients. High frequency ventilation has been found to have

retrospective study was to assess whether this clinical impression could be confirmed.

Patients and methods

Inclusion criteria

Patients were included in the study if the following criteria were fulfilled:

- an age of 16–65 years
- an injury severity score > 17 points
- the primary admission was under the trauma service of Braunschweig Gen. Hospital or Hannover Med. School
- they were treated with continuous axial rotation for impaired lung function

Definitions

ARDS was diagnosed if all the following criteria were met:

- there was respiratory insufficiency ($\text{PaO}_2/\text{FiO}_2 < 150$)
- there were bilateral diffuse infiltrations on the chest X-ray
- the pulmonary capillary wedge pressure was < 18 mmHg
- the PEEP was > 8 cm H_2O

Sepsis was diagnosed when body temperature exceeded 38.5°C , and if the leucocyte count exceeded 15000 cells/ μl whole blood or if a sudden rise in leucocyte count was associated with a positive bacterial blood culture.

Multiple Organ Failure (MOF) was determined according to a MOF-score. The diagnosis of multiple organ failure was made when the MOF-Score exceeded six points on two consecutive days or if more than two internal organs had to be artificially supported. Septic multiple organ failure was diagnosed if positive blood cultures were obtained under the condition of organ failure. The severity of injury was assessed by injury severity score (ISS)¹⁴.

General principles of therapy

The principles of primary care of injured patients are comparable as there is a close clinical cooperation and exchange of medical staff. Early intubation and ventilation were performed. Fluid replacement was calculated from arterial pressure, central venous pressure, kidney function (urinary output > 80 ml/h) and the pulmonary capillary wedge pressure. Positive end-expiratory pressure was maintained between +6 and +15 mmHg. The inspired oxygen fraction (FiO_2) was adjusted to maintain arterial oxygen saturation above 90 per cent. The ratio of inspiration and expiration (I:E) was adjusted between 1:2 and 1:0.5 according to blood gas analysis. A pulmonary artery catheter (SWAN GANZ flow directed thermodilution catheter model, Criticath, Mod. SP 5107, Spectramed BV, Bithoven, Netherlands) was inserted in patients showing signs of respiratory distress. Cardiac output was determined

Table 1. Criteria for early prophylactic kinetic therapy

Severity of injury (ISS > 25 points) or
Thoracic trauma (AIS _{thorax} > 3 points, Lung contusion on initial chest X-ray) or
Horovitz-ratio ($\text{PaO}_2/\text{FiO}_2$) < 150 and
Fluid balance (I/O ratio) upon positioning or 1 day prior (> +4 l) or
Blood transfusion > 15 units/12 h after injury or
Decrease of thrombocyte count (> 40000 cells/ μl per 24 h)

by thermodilution technique. The mean of three consecutive measurements was calculated.

Principles of kinetic therapy

Kinetic positioning was performed using a specialized bed (KCI-Mediscus Inc., Hoechststadt, Germany) and was applied to patients at high risk of developing impaired pulmonary function. The parameters shown in Table 1 were used to specify these patients.

Group distribution

Group P: Early prophylactic positioning: Patients were included if at least two of the criteria listed in Table 1 applied and if positioning was performed prior to deterioration of oxygenation.

Group L: Late positioning: Patients were included if impaired oxygenation was present ($\text{PaO}_2/\text{FiO}_2 < 150$) when kinetic therapy was initiated.

Criteria of subgroups

Two subgroups were studied: Groups P_{surv} and L_{surv} . These subgroups contained only *surviving* patients who *responded to therapy* and who had *impaired lung*. They were selected from the groups P and L respectively.

Time schedule

All data were documented from the time of admission to the trauma intensive care unit (TICU) and were continued on a daily basis until the patient was discharged from the unit. All data were collected at the same time each day (8:00 a.m.) from the patient's chart. In the figures time 'A' represents admission to the TICU and time 'B' marks the beginning of continuous axial rotation.

Principles of continuous rotation therapy

We have used axial rotation for respiratory insufficiency since 1989. After testing both axial rotation and intermittent prone and supine positioning we have preferred to use the former which had the following advantages:

- (1) The degree of rotation can be selected separately for each side according to the individual severity of pulmonary dysfunction (unilateral lung contusion).
- (2) During therapy there appears to be less danger of dislocation of endotracheal or tracheostomy tubes and thoracic drains and other catheters (e.g. central venous/Swan Ganz).

Table II. Demographic data of both patient groups

Parameter	Group L	Group P	P-value
Number of patients	54	105	
Age (yr)	42.1 ± 3.6	46.2 ± 3.1	n.s.
ISS (points)	30.3 ± 5.1	33.6 ± 2.6	n.s.
AIS _{thorax} (points)	2.8 ± 1.4	3.0 ± 1.0	n.s.
Non-responder n(%)	13 (24.7%)	11 (10.4%)	<0.05
Sepsis n(%)	05 (9.3%)	09 (8.5%)	n.s.
MOF n(%)	09 (16.6%)	15 (14.3%)	n.s.
Death n(%)	22 (40.7%)	26 (24.8%)	<0.05
Mean survival (days)	11.7 ± 2.4	20.9 ± 3.9	<0.05

(3) In cases of concomitant head trauma elevation of the head may be maintained during rotation without interruption.

(4) Should traction be necessary this can be applied and maintained during rotation as in the supine position since the extremities are stabilized laterally.

Statistics

All data were tested at 95 percentile, significant differences were estimated at $P < 0.05$. Variables fitting nominal scaling were tested using the Chi-square test. If variables corresponded to ordinal scaling, the Wilcoxon-test was applied for combined variables and the Mann-Whitney U-test for uncombined variables. Data with a normal distribution and rational scaling were tested with the *t*-test. In Tables I-IV and Figures 1-3 data are given as the mean ± SEM (standard error of the mean). Values significantly different from the baseline (day of positioning, time (B)) are marked by (+), significant group differences are marked by (*).

Results

One hundred and sixty one multiply injured patients were submitted to continuous axial rotation between 1 February 1989 and 31 December 1996. Twenty seven patients were treated at the Braunschweig

Table III. Subgroup analysis of surviving patients with impairment of pulmonary function (prior or after initiation of kinetic therapy), who responded to rotation

Parameter	Group L _{surv}	Group P _{surv}	P-value
Number of patients	22	53	n.s.
Age	38.2 ± 6.6	42.7 ± 3.4	n.s.
ISS (points)	28.1 ± 5.12	32.9 ± 7.6	n.s.
AIS _{thorax} (points)	2.2 ± 1.1	2.4 ± 1.5	n.s.
ARDS	11 (54%)	8 (15.1%)	<0.05
Pneumonia	6 (27.2%)	07 (31.8%)	n.s.
Duration of kin. therapy (days)		13.9 ± 4.7	11.8 ± 3.3
Ventilation time (days)	27.2 ± 5.6	24.3 ± 5.1	n.s.
Intensive care time (days)	33.2 ± 5.8	31.7 ± 4.5	n.s.

(Group P_{surv}: within the group submitted to early prophylactic positioning, only surviving patients who responded to therapy and had impaired lung function were selected. Group L_{surv}: Within the group submitted to late prophylactic positioning, only surviving patients who responded to therapy and had impaired lung function were selected).

Table IV. Complications during the course of intensive care and causes of death

Parameter	Unit	Group L	Group P	P-value
Non-responder	n (%)	13 (24.1%)	11 (10.5%)	<0.05
ARDS	n (%)	40 (74.1%)	36 (34.4%)	<0.05
MOF	n (%)	09 (16.7%)	15 (14.3%)	n.s.
Death	n (%)	21 (38.8%)	24 (22.9%)	<0.05
Death from ARDS	n (%)	11 (20.3%)	09 (8.6%)	n.s.
Death from MOF	n (%)	05 (9.3%)	06 (5.7%)	n.s.
Death from HT	n (%)	04 (7.4%)	07 (6.7%)	n.s.
Death from shock	n (%)	01 (1.9%)	02 (1.9%)	n.s.
Mean survival (total)	Days	11.7 ± 2.4	20.9 ± 6.9	<0.05
Mean survival (ARDS)	Days	12.5 ± 3.8	14.5 ± 5.9	n.s.
Mean survival (MOF)	Days	13.2 ± 4	22.6 ± 12.1	n.s.
Mean survival (HT)	Days	7.5 ± 1.7	26.4 ± 13.9	n.s.
Mean survival (shock)	Days	0.5 ± 0.4	0.7 ± 0.5	n.s.

MOF = multiple organ failure; ARDS = adult respiratory distress syndrome; HT = head trauma; shock = traumatic hemorrhagic shock.

Hospital and 134 patients at Hannover Medical School. There were no statistically significant differences from the two hospitals in age, total injury severity and severity of thoracic injury (data not shown).

In two patients of group L continuous rotation had to be reinstituted for deterioration of oxygenation after an improvement had been achieved initially. These patients were excluded from the study, thereby leaving 159 patients for evaluation.

Demographic data are listed in Table II. In group L a significantly higher number of 'nonresponders' was found. There were no significant differences regarding age, total injury severity, severity of thoracic injury and the incidence of MOF between groups L and P.

Figure 1 demonstrates changes of oxygenation based on the calculations of PaO₂/FiO₂ (Horowitz) for groups L and P. Until day 17 after the beginning of positioning significant differences between both groups were measured. In group L the PaO₂/FiO₂ ratio was significantly worse at the time of positioning (time B) compared with day 1 after trauma. In group P oxygenation at the start of positioning did not change significantly compared with enrolment in the study or compared with day 1 after trauma.

Group L showed a significantly higher incidence of ARDS than group P. The incidence of pneumonia was comparable in both groups (Figure 2). Figure 3 depicts the duration of positioning and of the duration of intensive care therapy. There were no statistically significant differences.

The subgroup analysis of surviving patients with impairment of pulmonary function, who responded to rotation (group P_{surv} and group L_{surv}) is listed in Table III.

Discussion

The apparatus for continuous axial rotation was developed to prevent deep venous thrombosis in para- and tetraplegic patients^{11,17}. Subsequently it was discovered that kinetic therapy mobilizes mucus in

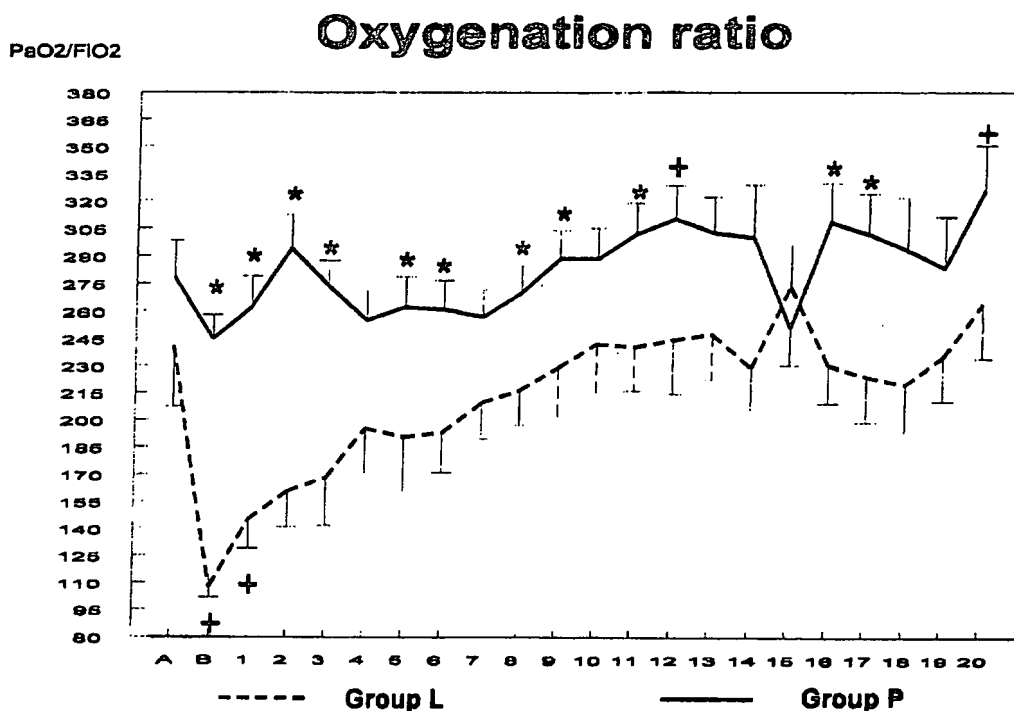


Figure 1. Course of the changes of oxygenation indicated by the oxygenation ratio (PaO₂/FiO₂).

artificially ventilated patients and thereby reduces pulmonary infections. It has also been shown to reduce the duration of artificial ventilation for acutely ill patients^{19,5,20,4,14}.

More recently, continuous rotation has been used to treat pulmonary complications in trauma patients. In these cases, pulmonary function may be altered by several mechanisms:

- haemorrhage and hypoxemia induce micro-vascular damage to pulmonary capillaries which leads to interstitial edema and ARDS,

- the lung acts as a 'filter' for emboli of fat from the fracture site and other debris from open wounds, and

- alteration of lung function may occur as a result of blunt thoracic trauma and lung contusion.

In recent years there have been increasing numbers of reports on the use of continuous axial rotation for improvement of oxygenation in trauma patients with pulmonary insufficiency and ARDS^{11,12,15}. It has been widely accepted that rotational therapy exerts positive effects on respira-

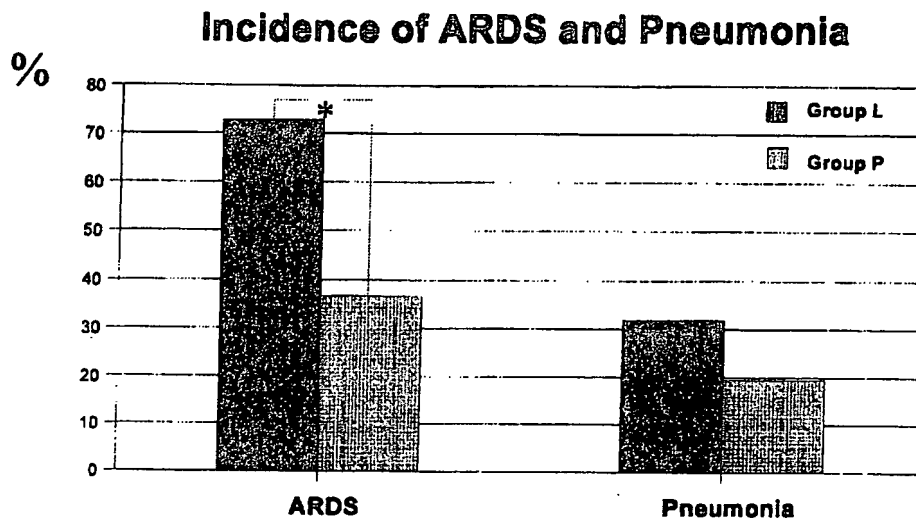


Figure 2. Incidence of ARDS and pneumonia in groups P and L.

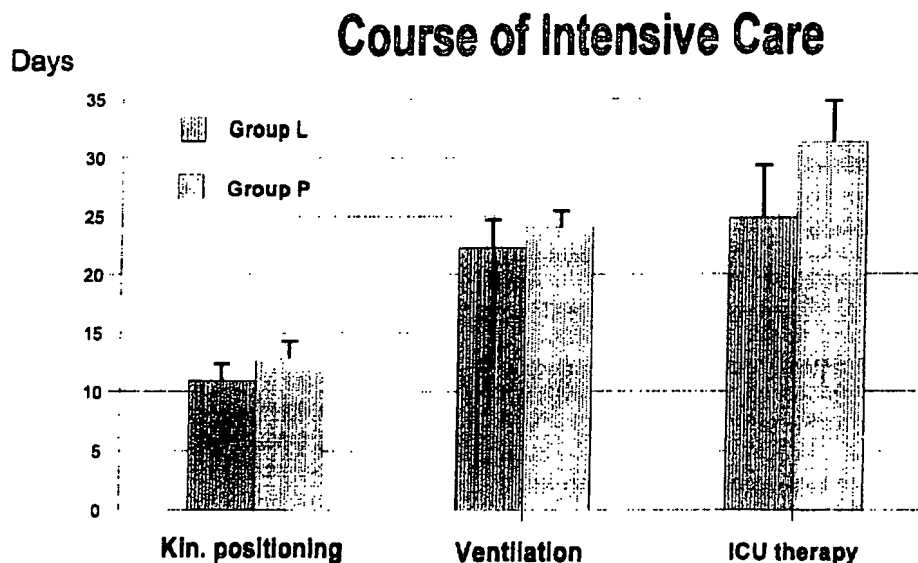


Figure 3. Durations of positioning, of ventilatory support and of intensive care in comparison between groups P and L.

tory function, comparable with alternating prone and supine positioning^{9,11,12,15}.

However, if axial rotation was begun after pulmonary function had deteriorated, in a considerable number of patients improvement of oxygenation was absent or not as profound as expected ('non responders'). Therefore, in 1993, we began to test the effects of early axial rotation which was initiated prior to deterioration of lung function. This was performed in patients with a high risk of ARDS (severe haemorrhagic shock, chest trauma etc.).

This concept can be criticized since some patients may not have developed respiratory distress even without axial rotation, i.e. kinetic therapy was unnecessary. In addition, unjustified costs may be incurred, if (unlike reports of other patient groups^{20,9,15}) axial rotation did not reduce the duration of ventilation. It was therefore the aim of the present study to investigate the following questions:

- Does early 'prophylactic' kinetic positioning in polytrauma patients reduce the incidence and mortality of pulmonary complications and ARDS?
- Does early kinetic positioning in polytrauma patients reduce the incidence of patients who fail to respond to this treatment?

The retrospective, non randomised design of this investigation is a drawback. We tried to compensate for this by collecting a large number of patients and by stratifying the groups to facilitate comparison. The groups were equivalent with respect to the degree of total and thoracic injury and the therapeutic interventions performed. The principles of ventilatory support did not change during the period of investigation in either hospital, furthermore, there was no change in the incidence of ARDS during the study period.

The results demonstrate a significantly lower incidence of ARDS in the group submitted to early treatment (18.1 per cent) than in the group submitted to late treatment (61.1 per cent). Furthermore, in the patients who did develop ARDS there was a lower mortality in group P (8.6 per cent) than group L (18.5 per cent), but this difference was not significant. Moreover, a significantly higher rate of non-responders and a significantly shorter duration of survival in succumbing patients was found in group L.

Figure 3 demonstrates a trend towards an of increased duration of kinetic therapy, ventilation and intensive care in group P. This effect may be secondary to a prolongation of life in patients who later succumb to their injuries. In fact, i.e. regardless of the cause of death, survival time in patients who subsequently died was longer in group P (20.9 days, group L: 11.7 days, $P < 0.05$). However, the mean survival time in patients who died from ARDS was about 2 days longer in group P (14.5 days) than in group L (12.4 days) (n.s.). As shown in Table IV, there may have been an influence on the duration of survival of patients who died of MOF and head trauma rather than of ARDS.

These results may be doubted since the influence of several factors have not been considered. First, some patients might have received axial rotation who did not require it. Second, in both groups a small number of patients were included whose pulmonary function did not respond to kinetic therapy. Third, some patients who died shortly after injury may have had an inappropriate influence on the results. In order to eliminate these effects, the subgroups L_{surv} and P_{surv} were selected.

In these subgroups comparison of the duration of positioning, ventilation and intensive care therapy was not statistically different, but showed a trend

towards shorter intensive care stay in group P.ⁱⁱⁱ While early prophylactic ventilation might have been expected to cause a prolongation of ventilation (and predispose to further complications) this effect was not found by our data. In contrast, a significantly lower incidence of ARDS occurred in group P.ⁱⁱⁱ despite a trend towards a higher total injury severity. In summary, the positive effect of early kinetic positioning was confirmed in analysis of these subgroups. The causes of the improvement in outcome after early axial rotation are not well understood, however, several ideas have been proposed:

- (1) The general effect of axial rotation in respiratory insufficiency appears to be a redistribution and mobilization not only of intrabronchial fluid (mucus) but also of interstitial fluid from dependent to nondependent parenchymal areas^{8,7,10}. It has been suggested that this might improve the ventilation to perfusion ratio and subsequently cause a reduction of intrapulmonary shunting^{11,12,16}.
- (2) It is possible that early axial rotation inhibits the development of dependant lung areas and thereby reduces atelectasis. Clinically, this effect may be clinically more relevant than microvascular injury associated with blunt trauma and haemorrhage.
- (3) Early positioning may also improve the total lung lymph flow.
- (4) In the presence of concomitant pulmonary contusion a significant reduction of the recruitable surface area of oxygenation has to be assumed even in the absence of initial signs on chest X-ray. A reduction of areas of dependant lung may add to the protection of pulmonary sites not involved in the sequelae of contusion. This effect may augment lung capacity which would not be available if the patient underwent positioning alone.

The positive effect of axial rotation on the reduction of pulmonary infection is well established^{19,5,20,14}. Our results are comparable to those of previous publications despite the heterogeneity of the groups investigated. Fink et al.¹⁹ found a reduction in the requirement of ventilatory support and a reduced hospital stay with the use of kinetic positioning.

In summary, prophylactically applied kinetic therapy was associated with a significant decrease of the incidence of ARDS, a tendency of shorter ventilation time in ARDS patients and a significantly lower rate of patients not responding to treatment. Furthermore, early kinetic positioning does not cause a longer duration of the total positioning time in groups L and P. In the patient groups selected for survival and possible unnecessary positioning there was a trend towards shorter positioning therapy, ventilation time and improved survival, which was not statistically different. Therefore prophylactic axial rotation in multiply injured patients does appear useful for the improvement of pulmonary function

and survival. We have been encouraged by our results and feel that further investigation is justified.

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Comments of a Devil's Advocate¹

A. CHARLES BRYAN

In Dr. Mellin's presentation, the effect of body position on pulmonary mechanics was emphasized. These interrelationships merit further comment. In acute respiratory disease, the apparent pulmonary gas volume is characteristically low. Logically, therapeutic mechanical efforts are directed at increasing the lung volume by means of positive end-expiratory pressure or by the continuous application of negative pressure at the body surface. Unfortunately, these commendable efforts are directed at patients maintained in the supine position, a posture characteristically associated with marked reduction of the pulmonary gas volume.

The adverse consequences of reduced lung volume are exaggerated by regional maldistribution of ventilated gas. If the diaphragm is regarded as a thin membrane separating 2 fluids of markedly different density (figure 1), the supine position is clearly associated with a volume deficit in the dependent parts of the lung, in the presence of the large abdominal hydrostatic pressure column. As a consequence, any tendency toward airway closure in dependent portions will be enhanced.

A study by Froese and myself (1) demonstrated that mechanical ventilation of patients aggravates further the loss of dependent lung volume. This proved to be true despite the observation by Kaneko and associates (2) that in the supine position ventilation is directed primarily to dependent portions of the lung. This latter apparently contradictory observation has been attributed to the smaller radius of curvature in the dependent portion of the diaphragm. The latter circumstance, according to the La-

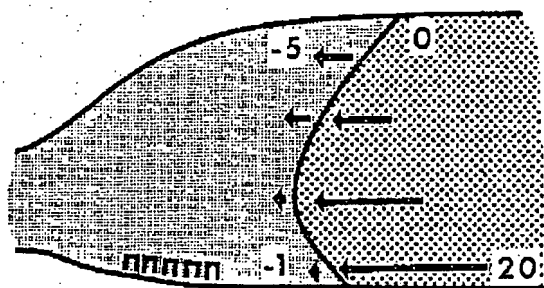


Fig. 1. In this supine model, the abdominal contents are represented as a liquid hydrostatic column exerting 20 cm H₂O pressure in the lowermost portion. This gradient is transmitted across the diaphragmatic barrier to the dependent region of the intrathoracic cavity. The mechanical consequences include a lesser gradient of pressure (-1 cm H₂O) in dependent portions of the lung, leading in turn to reduced inflation during inspiration.

place relationship, results in generation of a greater pressure for a given tension in the dependent region; the consequence is greater local volumetric displacement (figure 2). In contrast, a paralyzed, mechanically ventilated subject loses local mechanical advantage in the dependent region. The flaccid, passively shifted diaphragm assumes a different geometric configuration, and at the same time, relatively uniform pressure is applied to the entire thoracic surface of the diaphragm. Under these circumstances, the greatest diaphragmatic motion and volumetric displacement will occur where the opposing hydrostatic forces are minimal, at the top of the hydrostatic column in the nondependent portion of the lung. While the paralyzed, mechanically ventilated patient represents the extreme case, similar consequences attend mechanical ventilation of any patient. With efficient application of a patient-triggered ventila-

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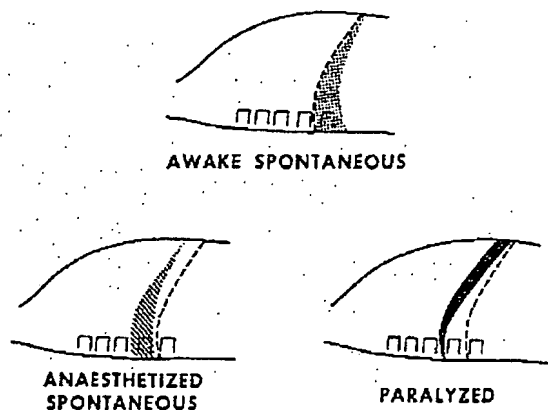


Fig. 2. The upper drawing illustrates diaphragmatic motion when lung inflation is achieved by voluntary effort. Motion and inflation occur predominantly in dependent portions of the lung. In contrast, during anesthesia and/or paralysis (lower drawings), motion and pulmonary inflation are distributed more evenly.

tor, a minimal pressure gradient is required to initiate inspiration. While initiation of inspiration is mediated through voluntary diaphragmatic activity, the remaining major portion of inspiration is passive, as in the paralyzed patient, and most inspired gas will flow to nondependent parts of the lung.

The position of the diaphragm in a paralyzed subject exposed to positive intrapulmonary airway pressure is such that the dependent portion of the lung cannot be expanded fully (figure 3). Use of positive end-expiratory pressure is ineffective because the lung will expand preferentially in nondependent portions where opposing forces are minimal. Perhaps, the only feasible means of expanding dependent portions of the lung is by placement of the body in such a position that ventilation of the normally dependent portions is facilitated. The prone position appears best suited for this purpose and merits serious consideration. Although logistic problems

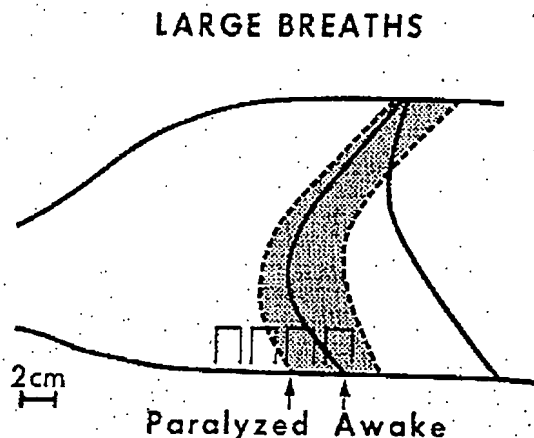


Fig. 3. In this supine model, the shift of diaphragmatic position during paralysis is depicted. Note that diaphragmatic motion during a large breath is greatest in the dependent region in the awake subject, but is more evenly distributed in the paralyzed state.

in adults would be considerable, compared to application in children, some type of orthopedic frame should provide a means of maintaining an inpatient in the prone position. By doing so, the adverse intrusion of intra-abdominal contents upon thoracic volume can be eliminated.

Summary of Discussion Pertinent to the Presentation by Dr. Bryan

It was noted that the prone position offered little or no gravitational advantage in facilitating drainage from the trachea.

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